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The Halford Oration.[1]

THE GOLDEN AGE OF MEDICINE.

By LESLIE HURLEY, M.D., M.S., F.R.A.C.P.,
Melbourne.

THE Halford oration, which the University has done me the honour of asking me to deliver, was founded to commemorate the life and work of George Britten Halford, first professor and later first dean of the faculty of the Melbourne Medical School. He was entrusted with the very difficult and responsible task of establishing and guiding the destinies of the school in its early formative years.

The University of Melbourne was opened in 1855, although the foundation stone had been laid in the previous year. Two years later Dr. Brownless induced the Council to resolve to "take such measures for the establishment of a Medical Faculty as the finances of the institution and the circumstances will admit".

In 1861 the Chancellor, Sir Redmond Barry, protested that "notwithstanding the renewed efforts made by the Council to establish a Medical School they had met with further obstacles".

In 1862 the Council, with much courage and enterprise, determined to make an attempt to launch the school. Finance was obtained partly by a Government grant and partly by economizing in various directions. Deductions were made in expenditure on the grounds and in the salaries of the lecturers in law and engineering, who were said by the Chancellor to have "cheerfully acquiesced". At any rate they allowed themselves to be operated on without any anæsthetic for the benefit of the medical profession.

In Sydney, on the other hand, the establishment of a medical school was delayed partly by the protests made by the professors on the grounds that the Medical School would delay the completion of the curriculum in the Faculty of Arts.

The Melbourne Medical School thus became the first of its kind to be established in Australasia.

The responsibility of choosing a man to undertake the difficult task of establishing the school was very wisely entrusted to two of the ablest and best known medical men in Great Britain, James Paget and Richard Owen, both of whom were subsequently knighted for their contributions to medical science.

After very careful searching and serious deliberation they chose Halford, whom they described as "one of the most distinguished physiologists of the day—a name that would give distinction to any university".

Halford arrived in Melbourne on December 22, 1862, accompanied by his wife, four daughters and a baby boy born on the voyage. There were as yet no buildings in which to carry on the work of the Medical School, so lectures were commenced on May 1, 1863, in a shed at the back of Halford's house in Madeline Street (now Swanston Street), to a class of three second-year students.

Halford set to work energetically, and the buildings of the Medical School, which remained substantially unaltered until comparatively recent times, were completed in 1865.

[1] The seventeenth Halford Oration, delivered at the University of Melbourne on June 18, 1951.

In addition to giving lectures in anatomy, physiology and pathology, Halford had the responsibility of establishing and administering the infant Medical School. Although Paget had recommended a course of study of three, or at the most four, years, it was wisely decided to make it a five years' course. The proficiency of our medical graduates and the high regard in which they are held abroad testify to the soundness of the foundations which he laid, and to the high standards which he set.

Halford was also keenly interested in research, and despite his many and varied duties found time to play his part in enlarging the boundaries of human knowledge. While still a student he did some original work on the mechanism of the heart's action, and later on made valuable contributions on the heart sounds, on the pericardium, on the comparative anatomy of the hand and foot in man and monkey, and on snake venoms. However, the time was not ripe, nor were the facilities and support available, for the establishment of a school of research.

Eminent medical men from other parts of the world have frequently commented on the discrepancy between the efficiency of our medical graduates and the very meagre output of research from our Medical School.

When we consider—despite the great difficulties with which he had to contend and the small resources at his command—that Halford was able to create a very efficient school for the training of medical men, it should not be impossible for us to establish in association with the Medical School a first-class research centre, where not only our own graduates but medical men from over the seas may be given an opportunity of searching after truth for truth's sake, unfettered by any limitations of immediate practical gains. If our Medical School continues to remain largely a teaching body, it will sooner or later lose the high regard with which it is held in other parts of the world. We have the men, as is amply shown by the eminence obtained in the field of research by Australians both here and abroad. What are needed, are the courage, determination and enterprise that were exemplified by the early pioneers, who succeeded in establishing a university in 1855, less than twenty years after Melbourne was virgin bush inhabited by only a few nomadic tribes.

While searching for a title for this oration I happened to read an article in an overseas medical journal, which, after describing the advances made in medical science during the present century, made some very adverse and disparaging comments about the state of medicine in the year 1900. Indeed, anyone unacquainted with the history of medicine would have concluded that medicine as a science began with the present century. It was quite definitely asserted that the present age was the Golden Age of medicine. Sitting on the banks of a beautiful mountain stream, which had been giving forth its sweet, soft music for countless generations, one wondered whether the stream of medicine could have had such recent beginnings.

It is quite true that at the end of the nineteenth century there were few diseases for which we had specific remedies, and many of these had been discovered largely in an empirical fashion. Medicine was still chiefly descriptive and treatment mainly symptomatic and expectant. During the past fifty years, owing to the rapid development and application of new scientific knowledge to the prevention, diagnosis and treatment of disease, the practice of medicine has been revolutionized, and we now have a specific therapy which often strikes at the cause for many diseases which previously were treated expectantly and symptomatically. Pneumonia, typhoid, typhus, pernicious anæmia, dysentery, and bacterial and coccal meningitis are merely examples of many diseases for which, since the dawn of the present century, we have for the first time an effective and rational mode of therapy.

The physician is even invading the domain of the surgeon. Some varieties of toxic goitre can now be treated effectively by medical means, and since the advent of chemotherapy and antibiotics, operations for empyema and mastoid infection are much less frequently performed than before.

When one considers what can be done medically for a patient now, compared with what could be done fifty years ago, there would seem at first sight to be no doubt that the present age can rightly and justly claim to be "the Golden Age of medicine". Before we concede this claim, it would be but common justice to examine the claims of preceding ages, to consider the natural tendency of each age to magnify its own achievements, and to make due allowance for the fact that each generation has the advantage of the accumulated knowledge and experience of all preceding generations.

The extent to which an age may magnify and glorify its own achievements is exemplified by the statement made by the great Ambroise Paré, the father of modern surgery. He considered he had raised the standard of surgery to such a high level of efficiency that there was little left for his successors to discover; and he was a humble man, as is shown by his oft-repeated statement, "I dressed him and God healed him". Within the boundaries of knowledge as it then existed, he introduced improvements of a fundamental and far-reaching character. It was he who invented artery forceps, reintroduced the use of the ligature, and showed that wounds could heal by first intention if dressed with a simple non-irritating substance and not with "oil of elders scalding hot". He did not see—nor perhaps, considering the limitations of the age in which he lived, could he have been expected to see—"that each little discovery pushes the curtain back a little further, but infinity lies beyond".

May we not be committing a similar error? May not our achievements seem small when viewed in the sober light of history by some future generation?

There are still many known fields to conquer (for example, cancer), and perhaps many heights as yet beyond the range of our vision, which have not yet even been seen, much less scaled.

As the world grows older each generation owes an increasing debt to the past. Each age has the accumulated knowledge and wisdom of preceding ages to light the way and serve as a guide to the further advancement of knowledge.

Consider for a moment the history of the development of our knowledge concerning the true meaning and physiology of the process of respiration. Many other examples could be taken, but this will serve to illustrate the point.

Before Harvey's time it was thought, as had been taught by Galen, that the object of respiration was to cool the fiery heart and to introduce air for the generation of vital spirits in the left ventricle. Harvey, the discoverer of the circulation, showed that blood is changed from dark venous to bright arterial in the lungs, but further than this he did not go.

Boyle demonstrated that air was necessary for life as well as for combustion.

Robert Hooke showed that by the blowing of a bellows over the open thorax of a dog the animal could be kept alive, thus showing that the essential feature of respiration was not in the movement of the chest but in certain blood changes in the lungs.

Mayow demonstrated that dark venous blood is changed to bright red by the taking up of a certain ingredient from the air.

Carbon dioxide was discovered by Black, hydrogen by Cavendish, and nitrogen by Rutherford.

Oxygen was isolated by Priestly and Scheele. Priestly came very near to solving the problem when he noticed that vitiated air could be renewed by vegetating plants, but being a confirmed believer in phlogistin he regarded respiration as being the phlogistication of dephlogisticated air.

It was left to the genius of Lavoisier, about a century after Harvey's great discovery, to demonstrate the exact nature of the interchange of gases in the lungs. Unfortunately, Lavoisier, while still at the zenith of his power, was wantonly and senselessly sacrificed to "*Madame Guillotine*" during the terror of the French Revolution.

Without in any way detracting from the brilliant work of Lavoisier, this very brief survey serves to illustrate that it is often very misleading to attribute any great medical discovery to a particular research worker at some given period of time. Every investigator, especially in modern times, derives knowledge, ideas and inspiration not only from his predecessor but also from his colleagues and co-workers.

Most of the credit goes to the individual who converts a theory, no matter how well supported by circumstantial evidence, into a proven fact, and particularly if he also develops it for the benefit and service of mankind.

If one traces the history of other great medical discoveries it will usually be found that many years, even centuries, before, the path has been prepared by men whose name have very often passed into undeserved oblivion.

We should be very careful, therefore, before we, too, lightly assume that the great advances made in scientific medicine in the past fifty years have been due entirely, or even largely, to the work of our contemporaries.

There is no greater corrective to conceit—and incidentally to fads and fashions in medicine—than a careful study of the history of the development of our art and science.

> A study of the history of medicine should form a part of our curriculum, not only because it is interesting but because it prevents undue magnification of our own achievements, serves to preserve a broad outlook and to foster humanistic and cultural ideals which are as much a part of our profession as its purely scientific aspects.

History, as it is so aptly expressed by Trevelyan, "is the cement which holds together all the studies relative to the nature and achievements of man".

Having briefly discussed the debt the present owes to the past, and the natural tendency of each age to glorify its own achievements, we may briefly travel along the path of history, survey the outstanding milestones, and attempt to make a critical judgement as to whether we are justified in regarding the half-century just passed as being the Golden Age of medicine, or whether some previous age has a greater claim to that honour.

Prehistoric man regarded all disease as being of supernatural origin and did not consider that it might spring from natural causes. He ascribed disease either to the presence of some evil spirit in the body of the patient which may have been projected there by his enemy—for example, in the pointing of the bone as practised by the Australian aboriginal today—or to the abstraction of the soul from the body by magical means. Cure was brought about by the medicine man's going into a trance and using appropriate spells and incantations, thereby hoping to remove the evil spirit or restore the wandering soul to the body.

Closely allied was the idea of casting out or transference of the disease from the patient to some other individual animal or plant. This conception has persisted into comparatively modern times—for example, in the supposed curative touch of a dead man's hands, particularly that of an executed criminal. The Royal touch was thought to be a cure for the King's evil even as late as Queen Anne's time, and Richard Wiseman, the leading surgeon in England in King Charles's reign, testified that he had personally witnessed hundreds of cures by this means.

Thus prehistoric man regarded disease as being due to magical causes, and treated it by methods not dissimilar to those employed by the psychiatrist today. Although we may be inclined to smile at his simplicity and ignorance, it is still true that much ill-health is due to psychological causes, a fact which is all too frequently overlooked in this age of science, specialism and dialectic materialism.

Let us now briefly survey the age which extended from the beginning of recorded history to the classical period of Greece—that is, from about 3000 B.C., when Shen Nung wrote the Great Herbal or Chinese *materia medica*, which is still in use in China today, to the time of Hippocrates, who was born about 460 B.C.

The earliest written records of medical practice came from the ancient civilizations of China, Egypt, Babylonia, India and Greece. A study of these records shows that surgery had made some progress. Abscesses were opened, dislocations reduced and fractures treated with splints. There is also evidence that operations of quite considerable magnitude—for example, lithotomy, Cæsarean section, excision of tumours and rhinoplasty—were performed by the ancient Hindus.

In Babylon, we get some indication of the degree to which surgery had developed by the following extract from a code of laws drawn up by one of the early kings:

> If a doctor treats a gentleman and opens an abscess with a bronze knife and shall preserve the eye of the patient he shall receive ten shekels of silver; on the other hand, if a doctor shall open an abscess with a bronze knife and shall kill the patient or shall destroy the sight of the eye his hands shall be cut off.

The Old Testament and the Talmud contain much information on personal and social hygiene, and might almost be regarded as the first text-books on public health.

In medicine, however, although a few simple drugs had been discovered, particularly by the Chinese and the Egyptians, disease was still regarded as being of supernatural origin.

The Ebers Papyrus shows that spirit and devil possession was the dominant idea in Egyptian medicine, and even when drugs were prescribed they were accompanied by the appropriate spells and incantations.

The Babylonians considered disease to be the work of demons, and treatment consisted mainly in an attempt to cast them out. Astrology and divination were largely used, not only in matters medical, but also in the common affairs of life. For divination, a model of a sheep's liver carefully mapped out into squares was used and compared with the liver of a freshly sacrificed animal. Any differences were noted and a deduction drawn from the result. Thus we read in Ezekiel xxi : 21: ". . . the king of Babylon stood at the parting of the way . . . he consulted with images, he looked in the liver."

Amongst the Hebrews disease was largely regarded as being a visitation from God, as is shown by the following passage from Exodus:

> If thou will diligently hearken unto the voice of the Lord thy God, and will give ear to his Commandments and keep his statutes, I will put none of these diseases upon thee which I have brought upon the Egyptians.

In ancient Greece, on the island of Delphi, the cult of temple healing was established. The patient slept in the temple, and during the night Æsculapius appeared as in a dream and gave advice, and in the morning the patient awakened cured. There were no failures. Snakes assisted in the treatment by licking the eyes and diseased parts of the patient. Later on physical treatment in the way of baths and exercises formed an important part of the cure.

Temple sleep as a means of cure was practised long into the Christian era, and even today relics of it still persist along the eastern Mediterranean littoral. Thus, before the advent of Hippocrates, although some progress had been made in surgery and a rudimentary system of personal and social hygiene had been developed by the Hebrews, medicine was still dominated by mysticism and superstition, and disease was regarded as being of supernatural origin.

Such was the state of internal medicine when Hippocrates, by common consent the greatest physician of all time, appeared on the scene. It was he who delivered medicine from superstition and taught that disease, even the divine disease epilepsy, was due to natural causes and was subject to natural laws which he attempted to formulate. For the first time in history he introduced into medicine a clear critical spirit of scientific inquiry. He also gave to it its high ethical ideal. As Garrison puts it: "It was the chief glory of Greek medicine to have introduced that spontaneous first-hand study of nature with a definitely honest intention, which is the motor power of modern science." It was Hippocrates who introduced and developed the

science of clinical medicine. By careful and accurate observation, recording of facts, separating essentials from non-essentials, he attempted to discover the principles and laws which govern the origin, course and termination of disease. Many of his descriptions of disease, with a few modifications, could take their place with credit in any modern text-book, and his accurate and honest descriptions of actual clinical cases, followed through to their termination in death or recovery, are almost the only record of their kind for the next 1700 years. He was the first to describe pleural friction and Hippocratic succussion, and the details of the facial appearance in the presence of impending dissolution, which is known even today as "the Hippocratic facies". Laennec freely acknowledged that he would not have discovered the stethoscope which every doctor carries today had he not got the idea from reading the works of Hippocrates.

The Hippocratic books on fractures, dislocations and wounds, when we take into consideration the limitations under which they were written, have been pronounced by eminent medical scholars to be the equal of any similar work today.

Hippocrates gave the first description of the healing of wounds by first and second intention, described the symptoms of suppuration, and insisted that in such cases medicated dressings if applied at all should be not in the wound but around it. If water was used for irrigation it should be pure or boiled, and the hands and nails of the surgeon should be cleansed. He thoroughly distrusted the value of treatment based on fanciful theories as to the cause of disease, and when he could not confirm the beneficial result of any particular treatment from his own actual observation, he preferred to rely on the healing powers of Nature rather than on meddlesome and often drastic and harmful methods of treatment. With no literature to refer to, and no guiding beacon from the past, and with no equipment other than a keen mind and acute powers of observation, he accomplished for internal medicine all that it was possible for human mortal to accomplish. Unfortunately, during succeeding ages many of the lessons which he taught were forgotten, and it was not until the Renaissance that they were relearned and put into practice again.

With the decline of Greece, and particularly after the fall of Corinth in 146 B.C., Greek medicine was transported to Alexandria and to Rome. It had, however, already lost some of its fire and lustre, and had become more interested in formalism and dogmatism than in observation and investigation.

All the leading physicians of the Roman Empire with the exception of Celsus were Greeks, and the best known of these was Galen (A.D. 131 to 201), whose views dominated European medicine for the next twelve centuries. During this period his pronouncements were accepted without question, and anybody who dared to differ from them was regarded as a heretic. Although many of his views were erroneous, he did make many substantial contributions to medicine. He was the first to describe the cranial nerves and the sympathetic nervous system. He also made experimental sections of the spinal cord, producing partial hemiplegia, and divided the recurrent laryngeal nerves, producing aphonia. In addition he gave a very good account of the classical signs of inflammation—redness, swelling, heat and pain. However, he was responsible for many errors, partly owing to the fact that his anatomy was based on dissection of pigs and apes, but more particularly to his tendency to subordinate fact to theory. He was, to some extent at least, the product and the victim of his age, which was one of dogma and theory rather than of science and logic. His most important errors were his belief that blood passed by invisible pores through the interventricular septum, his belief that suppuration was an essential process in the healing of wounds, and his doctrine of vitalism, which held that blood received natural spirits from the liver and vital spirits from the left ventricle, and that vital spirits were converted into animal spirits by the brain, the whole organism being animated by a hypothetical pneuma. Unfortunately his theories were accepted as facts, with the result that the progress of medicine was retarded for many generations.

After the fall of Rome came the dark ages. The preservation of scientific medicine passed into the hands of the Arabs, or rather of those who spoke Arabic and belonged to the great Arabian empire which extended from Spain to Samarkand. They translated the works of the Greeks into Arabic and made a few original observations of their own, particularly in pharmacology and chemistry. They invented methods of sublimation, distillation and crystallization. The word "drug" itself is of Arabic origin, and so are benzoin, camphor, senna, laudanum, alcohol and alkali. Their interest in chemistry may have been stimulated by the injunction of Mahommed: "O Servant of God use medicine because God hath not created pain without a remedy for it." They produced two very great physicians, Rhazes (A.D. 860 to 932), who separated measles from smallpox, and Avicenna (A.D. 980 to 1057), who wrote extensively and was the first to maintain that pulmonary tuberculosis was a contagious disease. His works are still used in the east, and as late as 1650 were prescribed as standard textbooks at Montpellier, where the great Sydenham studied after leaving Oxford.

In Europe, during the dark ages, the views of Galen were accepted without question. Learning was discouraged, dissection of the human body was practically forbidden, and it was dangerous to question authority or express original ideas. In many places medicine was largely controlled by the church. Once again, as in ancient Israel, disease was thought to be sent as a punishment for sin, and prayer and fasting were the sovereign remedies. For those who resisted the medical influence of the church, diagnosis and prognosis rested largely on the state of the stars and inspection of the urine. Treatment consisted mainly in blood-letting and the use of herbs, mostly inert.

Thus during the Middle Ages, although the Arabs kept alight the torch of Greek medicine and made a few minor contributions of their own, European medicine produced nothing to advance medical science, but actually retarded it.

After the Middle Ages came the Renaissance. There was a revival of the ancient culture of Greece and Rome. Men began to think, to question authority and to base their beliefs not on theory or dogma but on observation and experiment. The medical world returned again to the sound teachings and logical scientific methods so clearly laid down by Hippocrates. By the work of a few brilliant men the basic sciences of anatomy, physiology, pathology and medicine were soundly and firmly established.

First came Vesalius (1514 to 1564), the most brilliant star in the medical firmament since Galen. In his epoch-making work "*De fabrica humani corporis*" he completely revolutionized the science of anatomy. In it he disposed of many Galenical superstitions—for example, the possibility of blood passing through the interventricular septum, the five-lobed liver, the double bile duct, Adam's missing rib and the horned uterus. He may rightly be regarded as the greatest anatomist of all time. In the clinical field he was the first to diagnose and describe aneurysm of the abdominal and thoracic aorta, and he also operated successfully for cancer of the breast and empyema (on Don Carlos of Aragon).

Then came Harvey (1578 to 1657), who conclusively proved that the blood circulated, probably the most important medcial discovery up to this time; with it was born the modern science of physiology. One of the main points in Harvey's argument was that the actual quantity and velocity of the blood made it physically impossible for it to do otherwise than circulate. It was the first time that the idea of mathematical measurement was applied to the solution of a physiological problem. The method by which his discovery was made, by careful observation, well-planned experiments, and collection of facts followed by inductive proof, was just as important as the discovery itself. It has served as a model for future workers.

Following closely after Harvey, Sydenham (1624 to 1680) in England, and Boerhaave on the Continent, rescued medicine from the theorists and demonstrated once again that

accurate observation with careful sifting and correlation of facts was much more important than vague theories.

Two years after Harvey's death Morgagni was born. He laid the foundation of modern pathological anatomy. For the first time the history of the patient and the clinical findings were correlated with the findings in the body after death. Many people before him had examined diseased organs with a certain amount of intelligence, but Morgagni was the first to do this in a general and systematic manner.

Thus by the middle of the eighteenth century the basic sciences of anatomy, physiology, pathology and clinical medicine were placed on a sound foundation which has held firm ever since.

During the next hundred years, gradual but steady progress was made. More and more additions were made to the solid edifice which had already been erected by the men of the Renaissance. A detailed knowledge of the post-mortem appearances in most of the common diseases was rapidly acquired. Macroscopic and microscopic anatomy developed apace. John Hunter, although he made no outstanding discovery, made many observations which were of immense help to future workers. He described the transplantation of animal tissues, discovered the important principle of collateral circulation, and reintroduced proximal ligature for aneurysm.

Jenner demonstrated the value of vaccination against small-pox, although it was to be many years before viruses were discovered.

Considerable advances were made in clinical medicine by Heberden, Pott, Bright, Hodgkin, Addison and many others. Laennec discovered the stethoscope and made valuable contributions to pathology and physical diagnosis.

Important advances were made in physiology by Claude Bernard (1813 to 1878), who discovered the glycogenic function of the liver, the digestive function of the pancreas, and vasodilator and vasoconstrictor nerves. He showed that the different organs of the body did not function as isolated units, but were integrated and correlated by the action of the nervous system and internal secretions.

Virchow introduced the idea of cellular pathology, and in 1846 Morton popularized ether anæsthesia.

Despite the steady advances made in medicine and the allied sciences from the time of the Renaissance to the middle of the last century, we still knew little or nothing more about the cause and prevention of disease than did the ancient Greeks, although there was, of course, no lack of theories. We had learned, but only in part, to get rid of treatment based on theory and to trust Nature more and drugs less. A few valuable drugs had been discovered, mainly in an empirical fashion, but all too frequently drastic methods of treatment were used—for example, sweating, purging, bleeding and blistering—and many of the drugs prescribed were inert or useless.

However, the latter half of the nineteenth century was to see the sowing of the seeds of a rich harvest, the benefits of which we are reaping today. The rapid advances made in scientific medicine and the extraordinary accumulation of knowledge in the present century were made possible largely owing to the fundamental discoveries of a few men during the latter half of the nineteenth century. Of these the most outstanding was unquestionably Louis Pasteur, probably the greatest medical scientist since Hippocrates. It was he who showed that microorganisms were not just interesting little creatures to be viewed under a microscope, but that they played a most important part in Nature and in the affairs of man. He pointed the way not only to the causation but also to the prevention and treatment of the microbic diseases. He discovered and applied the principle of active immunization by using attenuated cultures.

It was Pasteur's proof that organisms existed in the air and were the cause of fermentation in milk and wine that led to the idea that wound infection might also be due to microorganisms. This was put to the practical test by Lister, with results that revolutionized the practice of surgery. He was even aware, as was recently pointed out by Florey, that certain air-borne organisms inhibited the growth of anthrax bacilli, and suggested that this phenomenon of antibiosis might be applied to the treatment of infection.

Then came Koch, who with Pasteur was the co-founder of the modern science of bacteriology. He proved that anthrax was due to the anthrax bacilli, which had been described thirty years before by Pollender and Davaine. He later on discovered the cause of tuberculosis and cholera; and during the next few years the causes of most of the common coccal and bacterial infections were determined.

Ehrlich introduced new methods of staining blood smears and laid the foundations of the modern science of hæmatology. He was also the first to set out to produce synthetically a drug for a specific purpose, and thus established chemotherapy on a firm basis. His laborious and brilliant researches, after some initial minor successes, culminated in the production of "Salvarsan" early in this century.

In 1885 Takaki proved the dietetic origin of beriberi and outlined measures which led to its eradication from the Japanese navy. Shortly afterwards Eijkmann produced beriberi experimentally in fowls, thus proving beyond all question the importance of accessory food factors.

Von Behring in 1890 discovered antitoxins and the principle of passive immunization.

Röntgen in 1893 discovered X rays. The significance of this discovery was at once recognized, and almost immediately applied to the diagnosis of fractures, bone diseases, renal calculi and similar conditions. Myxœdema, which had been described by Gull many years before, was successfully treated by thyreoid extract in 1891, the importance and practicability of hormone therapy being thus demonstrated.

In 1896 Ross proved that the anopheles mosquito was the vector of the malarial parasite and devised methods for the effective prevention of malaria, and in the same year Sir Almroth Wright and others introduced antityphoid inoculation, and thus established the principle that killed cultures of organisms were effective in the prevention of disease.

At the close of the century in 1898 the Curies isolated radium.

It is apparent, therefore, that although the treatment of disease at the beginning of this century was largely based on empiricism, the original and brilliant discoveries, the further development of which was responsible for the rapid advances in the twentieth century, had already been made, and their practical utility—it is true, only on a limited scale—had been demonstrated.

The causation of infection by living organisms, active immunization by attenuated and killed organisms, passive immunity, chemotherapy, X rays, radium, hormones, the importance of accessory food factors and even antibiotics, had all been discovered.

Huchard's statement that "the revival of Hippocratic methods in the seventeenth century and their triumphant vindication by the concerted scientific movement of the nineteenth, is the whole history of internal medicine" may not be an over-simplification of the truth.

The present century grasped this rich heritage with both hands, and proved itself not unworthy of the gifts it had received. There is no need to enumerate in detail the enormous practical utility of the advances which have been made. The sulphonamide drugs, penicillin and other antibiotics, the rapid extension of our knowledge of endocrinology, nutrition and vitamins, and the use of radium, X rays and radioactive isotopes in diagnosis, research and treatment, have revolutionized the practice of medicine.

It is important and not ungenerous to mention that many of these advances have been made possible by a tardy realization on the part of both governments and peoples that money spent on research is well spent and will pay handsome dividends.

Almost every branch of knowledge has its contribution to make to the tree of medicine, and if we are to progress

adequate facilities must be made available to those who have the ability and the enthusiasm to spend their lives in enlarging the boundaries of human knowledge. Although the lone investigator may still make his contribution, medical science has become so complicated and interrelated with other branches of knowledge that team work is now an essential for most major advances.

Apart from amplifying and developing to an extraordinary degree the work already begun in the last century, what new fields have we opened up for the next generation?

Viruses have been discovered and much new work can be expected in the future development of knowledge in this field.

The cause of cancer still remains a mystery; but recent work has given some hope that the problem may not prove to be insoluble. For the first time, at least one variety of cancer can be favourably influenced by treatment given to the host, rather than by a destructive attack on the tumour cells.

Essential hypertension, which has become almost "Public Enemy Number 1", can now be produced experimentally, and much valuable work has been done which gives some reason to hope for a future solution.

The discovery of cortisone and ACTH and their effect on rheumatoid arthritis, rheumatic fever and a number of diseases of unknown ætiology, for which we have as yet no effective therapy, may provide a means for their ultimate conquest. It has not yet been shown that these discoveries constitute a major advance in knowledge of the causation of disease or its treatment, although they may lead to something of importance in both of these directions.

Thus, no fundamental discoveries comparable in magnitude and importance to those made by Pasteur, Lister, Koch, Ehrlich, Röntgen and the Curies have been made in the past fifty years.

To what period, then, will future generations, viewing the past from the longer perspective of history, award the palm of being the Golden Age of medicine? We are perhaps too close to the present age to view it critically, but many recent medical writers have already awarded it that honour. If we view the past dispassionately and without prejudice, it would appear that there are four periods which merit serious consideration: the period of Hippocrates, when perhaps never before or since in the history of the world did so many men of genius appear together in such narrow limits of space and time; the Renaissance, which gave us Harvey, Vesalius and Morgagni; the latter half of the nineteenth century, which owed its inspiration largely to the genius of Louis Pasteur; and the present age.

Hippocrates created something out of nothing. With no assistance from the past, and by his own unaided efforts, he achieved a mighty revolution. Before him, nothing but abysmal darkness and superstition; with his advent, a shining light that brightens more and more as the years roll by. It was he who had laid down the solid foundation of the fundamental principles governing all scientific inquiry, on which medicine still builds today and will continue to build in the future.

From then onwards, and right through the Middle Ages, European medicine gradually reverted again to mysticism and superstition and became bogged down in a morass of dogma and theory. The foundations which had been erected by Hippocrates were allowed to rot and fall into disrepair. They were rebuilt and strengthened by the men of the Renaissance, when to question authority was dangerous.

From then onwards until the middle of the nineteenth century steady progress was made. Until the advent of Louis Pasteur, however, we knew little or nothing more about the cause and prevention of disease than did the ancients. It was through his work and inspiration, ably supported by Koch and Lister, that we came to understand the causation and the principles governing the prevention and treatment of wound infection, and of the great plagues which hitherto destroyed mankind.

The present age has, by a massed and concerted scientific attack, developed and brought to fruition for the benefit and service of man the fundamental discoveries made in the latter half of the nineteenth century.

To what age, then, shall we award the honour of being the Golden Age of medicine? If I were asked to give my vote I should cast it in favour of the age of Hippocrates, and my second preference would go to the age which produced Louis Pasteur.

A CLINICAL STUDY OF INFECTIONS OF THE NEWBORN OCCURRING IN A MATERNITY HOSPITAL OVER A SIX-MONTH PERIOD.

By CLAIR ISBISTER, M.R.A.C.P., D.C.H.,
Honorary Assistant Pædiatrician, The Royal North Shore Hospital of Sydney, Sydney.

> It is not yet possible to trace all forms of sickness to their source, for clinical medicine which portrays disease advances before clinical science which explains it; but experience suggests broad categories into which the injuries which produce disease in infancy may be placed (Spence, 1941).

Two of these categories form the basis for study in this investigation in the newborn period: (i) infections (the primary study) and (ii) temporary failure to establish the mechanisms of independent existence (less directly).

In the same article Professor Spence also states that "we may have to study the primary and infantile form of all infections, just as we have studied the illnesses produced by primary infection with tuberculosis".

It would seem a relatively simple matter to use Professor Spence's method of charting the infections occurring in the nurseries of an obstetric hospital over a six-month period (Spence and Farquar, 1946) and to observe their form and the factors that influenced them; and such I expected it to be, though I had the warning of Professor Spence and his colleagues (1949), Dr. D. Court (1949) and Dr. Mary Taylor (1949), who had done it in Newcastle and at Queen Charlotte's Maternity Hospital, that it was unpopular work, that every patient would have to be examined personally and daily during the investigation, and that no one else's findings could be accepted since the criteria of diagnosis might vary. It also appeared relatively simple, though time-consuming, to plan a bacteriological investigation in association with this, on a less detailed scale though of the same type as that carried out by Phyllis Rountree at King George V Memorial Hospital for Mothers and Babies (Rountree and Barbour, 1950), but to correlate the clinical and pathological investigations in order to get a clearer picture of the incidence of clinical disease in a non-epidemic period (Coventry and Isbister, 1951).

THE PROBLEMS OF DIAGNOSIS OF MINIMAL INFECTIONS.

However, as soon as the first part of the investigation was attempted, the truth of Professor Spence's warning became apparent, as did also the truth of the introductory statement made in the Report on Neonatal Mortality and Morbidity by the Committee of the Royal College of Obstetricians and Gynæcologists and the British Pædiatric Association (1949), namely:

> Infections of the newly born and the diseases they cause constitute one of the major problems of neonatal health and hygiene. Though these infective illnesses are frequent, doctors and nurses who undertake the care of newly born do not always recognize them and therefore fail to appreciate their significance.

The first month I spent keeping Spence charts, adopting, as far as my limited experience enabled me, the criteria of diagnosis used by Dr. Mary Taylor under Professor Moncrieff's supervision at Queen Charlotte's Maternity Hospital (1949), and establishing whenever possible patho-

logical proof of the existence of infection. This latter was essential as the whole subject obviously appeared to many a new and not very important field, and to both resident medical and nursing staff a "sticky eye" or a few pustules must surely be an anticlimax after the high drama of the labour ward.

I found that without definite clinical descriptions of conditions and without pathological proof, "prickly heat" and staphylococcal rashes, "separating cords" and umbilical sepsis, "silver nitrate reactions" and conjunctivitis, "milky mouth" and thrush, became identical, indistinguishable pairs of twins.

As further proof of this failure to recognize the infections of the newborn it is interesting to observe 108 consecutive admissions to a mothercraft home between January 1 and June 30, 1950. Every baby was accompanied by a doctor's certificate stating it to be free of infection, and nearly all came direct from a maternity hospital, yet there were 44 clinical infections found amongst these babies when examined by the visiting physician. As the nursing staff of this particularly home are extremely alert to possible infection and the nursing standard is high, immediate isolation is practised and cross-infection is almost absent. The matron is also empowered to refuse admission to babies with unquestionable thrush, and those refused admission in the period under consideration are not included in this number. There seems little doubt that the infections were present, in the majority of cases, when the doctor issuing the certificate examined the baby. I therefore feel it necessary to describe briefly the main clinical infections of the newborn that figure in this investigation.

In the six-month period under discussion there were no deaths with infection as the primary cause, though it figured in two as a contributory cause: (i) a one-pound-eight-ounce premature baby died at the age of one week of inhalation pneumonia and had an offensive cord; (ii) a baby with a clinical picture that fitted Craig's description of intracranial hæmorrhage (Craig, 1938) vegetated for three weeks and died with multiple ulcers in the intestine. Baby 14 (Figure III) provided the one instance of "probable septicæmia" with umbilical infection that ended in recovery, and in all other cases the infants had moderate to minimal infections. Post-mortem examinations were carried out after 24 of the 21 deaths and 24 stillbirths occurring during the period.

DESCRIPTION OF INFECTIONS OF NEWBORN.

"Sticky Eye."

The term "sticky eye" is applied to simple conjunctivitis with infection of the conjunctiva and purulent discharge sufficient to mat the lashes and render the eye sticky. No cases were included in which the condition responded to swabbing with boracic lotion and did not recur after several hours. In most of the cases in which culture was carried out hæmolytic *Staphylococcus aureus* was grown. Unfortunately for the figures, the interests of babies always took precedence over the cause of science, and treatment was often started before the baby was examined by me; material for cultures was therefore taken only if treatment other than with boracic lotion had not started. Of 79 cases there were only 28 in which swabbings were submitted to culture, with the following results: hæmolytic staphylococcus 20, *Bacterium coli communis* 2, *Staphylococcus albus* 2, *Streptococcus viridans* and *Staphylococcus albus* 2, sterile 1, no pathogens 1. It was routine to use silver nitrate in 0·5% solution in the eyes at birth, and most of the cases occurring in the general nursery had been called silver nitrate reactions. I personally have no doubt that the chemical irritant and the temporarily lowered resistance of the conjunctiva from its use enabled the infecting organism to become established; but these are bacterial infections.

Of the 69 cases of "sticky eye" occurring in this investigation, it is interesting to note the day of first appearance of infection and the different incidence in the annexe to the obstetric block from that in the other nurseries (see Figure I). The general nurseries and premature ward had a maximum incidence on the second day, when 10 of the 33 cases occurred—that is, 11 in the first forty-eight hours (one-third)—whereas in the annexe the maximum incidence was on the seventh day of life, 24 of the 36 cases occurring on the sixth, seventh and eighth day of life (two-thirds). Patients were transferred to the annexe on or after the fourth day, and as the infection rate was by then falling in the main block it appears that some factor was operating in this nursery that increased eye infections but no other infection. The nursery care was the same and the method of cleaning the nursery the same as in the other nurseries (namely, washing, not sweeping, the floor), but it is beside the main road, whereas the general obstetric block is pleasantly situated away from the road and surrounded by lawns and gardens, the atmosphere being much less dusty.

When the "sticky eye" and pustular rash charts are compared, it is apparent that in the main block the pustular rashes exceed the "sticky eyes" in number, as has been observed in hospitals with low infection rates—for example, Belfast (Frazer, 1948)—so that in the annexe the rise in number of eye infections is partially responsible for what was to me an unexpectedly high infection rate (57 of the 69 cases had occurred by the eighth day of life).

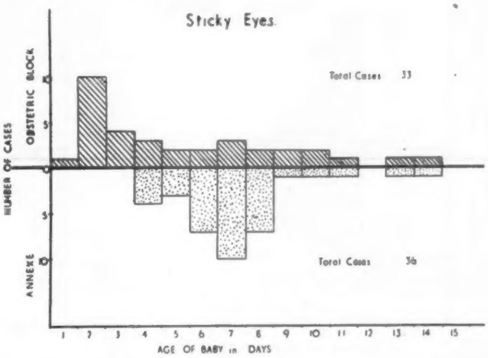

FIGURE I.

Chart illustrating the day of onset of infection in 69 cases of sticky eye occurring in the main obstetric block and the annexe to the obstetric block.

In this series no cases of inclusion conjunctivitis were recognized or sought bacteriologically, but I do not think any were missed as only three patients were affected for five days or more; one had inverted lashes and developed a corneal ulcer, another had a hæmolytic *Staphylococcus pyogenes* rash, and hæmolytic *Staphylococcus aureus* was grown from the profuse purulent discharge from the eye of the third.

Our cases did not fit into Dr. Campbell's classification (Campbell, 1951) in that rhinitis, *otitis media* and pneumonia were not associated with "sticky eyes", though it is obvious that such a sequence must be possible and occur in cases in which early treatment is not given. This difference may serve to stress again that the infections recorded in this series are minimal.

Skin Infections.

Pustular Rash.

The acute pustular rash caused by *Staphylococcus pyogenes* is a definite clinical entity, which I have seen only in the newborn period; it somewhat resembles impetigo. The first lesions are blisters with little surrounding erythematous area, and vary in size from a pin's head to the large bullous lesions of pemphigus; they rapidly fill with purulent secretion and may spread all over the skin in a few hours. They most commonly occur first behind the ears, round the neck and eyelids, and in the axillæ, and then spread over the body; most cases occurred in the first week of life. As a baby was considered one day old after 12 midnight, even when born ten minutes before, and as all calculations were made on its birth day,

it is possible that had these been calculated in hours we might have had an even narrower range of maximum incidence and possibly have thrown more light on the incubation period of *Staphylococcus pyogenes*. Charting of pustular rashes revealed a maximum incidence on the fourth and fifth days, whatever nursery the baby occupied; 64 of the 73 cases had occurred by the eighth day, and 41 of the 73 on the fourth or fifth day (see Figure II).

There was only one case of "bullous impetigo" or "bullous pemphigus" seen in this period, and that occurred in a baby after discharge from hospital. A less rapidly spreading form of this rash than the type described above is commonly seen towards the end of the first week and later; there is some inflammatory reaction round the blisters, and the commoner sites are round the umbilicus and on the inner sides of the thighs, particularly after circumcision, and on the buttocks. "Prickly heat" often predisposes to it. There were not many of these cases in our series, probably because the majority of patients were discharged from hospital by the tenth day, few circumcisions were performed, and the prickly heat was not particularly in evidence.

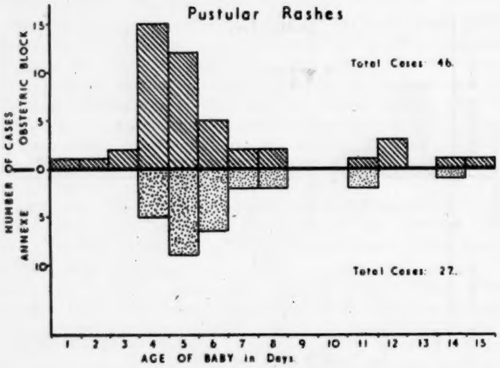

FIGURE II.

Chart illustrating the day of onset of infection in 73 cases of pustular rashes.

However, at the time of writing (February-March) there has been a sudden increase in the incidence of pustules, whereas for months we had had difficulty in finding a case for the students. The humidity and external temperature have been producing ideal incubator conditions, and in addition we have had a period of overcrowding such as we did not have in the period under review. The Spence chart for December, 1949, showed the decreased incidence of infection when the agitation to "be home for Christmas" reduced the numbers in the nursery.

Localized Abscess and Adenitis.

Localized abscesses and adenitis were very rare; they occurred in the second week or later, and there were only four in this series—two in operation wounds with little general reaction, one in a heel of a baby that had had several pricks over a week before, and the fourth a localized adenitis with no apparent cause. It appears that in the first week of life the baby has little power to localize infection.

Paronychia.

Paronychia appeared as localized redness round the nail bed, was non-suppurative, and was always associated with peeling and broken skin round the nail. Only four cases were observed, owing primarily, I feel, to the alert nursing staff's prophylactic treatment. No cultures were prepared from these cases, so that it cannot be said whether the infection was monilial or staphylococcal. However, all our cases were associated with either conjunctivitis or pustules—that is, staphylococcal infections—and we did not observe the association with infected buttocks and enteritis (Campbell, 1951) either in this series or in the Carpenter House series. It seems reasonable to assume that in hospitals with endemic thrush such an association would occur almost inevitably.

Thrush.

"White mouth" or "milky mouth" is due to *Monilia albicans*, a common invader of the vaginal mucous membrane in the pregnant woman, and therefore an ever-present risk to the baby in a maternity hospital; the organism being spore-bearing, the infection may be endemic in blankets, dust *et cetera*. The lesions in the mouth consist of white patches seen on cheeks, lips, tongue and palate, at first white and discrete, later greyish and confluent. They are not easy to wipe off and if wiped off leave an abraded surface. There is usually some little erythema around the lesions. Milk curds are easily wiped off and not usually found firmly lodged between gum margins and lips.

There were no cases of "thrush buttocks" or enteritis in our series; this is to be ascribed to the early diagnosis and prompt treatment by a "thrush conscious" nursery sister. This was in great contrast to the situation in Carpenter House, where in several cases the baby presented for admission with the raw weeping areas occurring in the napkin area. One thrush mouth associated with thrush buttocks had the appearance of tongue and cheeks being almost iced. (This occurred in a doctor's child, the indignant parent having been assured by the senior resident medical officer of the maternity hospital that the condition was definitely not thrush; the embarrassing refusal to admit the child to Carpenter House was fortunately supported by a positive slide examination result and a profuse growth of monilia. Two days' adequate treatment was followed by rapid recovery and ultimate admission.[1])

Several cases were observed in which the mother had cracked nipples with the typical white flecks of monilial infection. I assume that the monilial infection of the nipple is secondary both to cracked nipple and to thrush mouth in the baby, and is very rare. In this series there were 17 cases of thrush in seven months during which 1085 babies were born (1·5%). Unfortunately, there were not many cultures prepared, and in most cases the diagnosis was determined on clinical grounds. One mother had acute monilial vaginitis, and monilia was grown from a vaginal swabbing of another.

It is of interest that amongst the whole 17 there was not one normal healthy baby born after a normal labour: seven were premature; five were born after long and difficult labours, and three of these subsequently needed resuscitation; one had impacted shoulders and a fractured humerus; one had a precipitate birth; two were born by Cæsarean section; one was illegitimate and intended for adoption, had pustules and infected eyes, and was fully artificially fed. Of the 17, only two were fully breast fed and eight were artificially fed. Though the number is far too small to allow generalization, there seems little doubt that lowered resistance, either general or local, was a factor in these cases in initiating infection. An overwhelming dose of infection certainly does not appear to have been present, though it was possibly a factor in some cases among infants presenting at Carpenter House for admission.

I am sure that the infection is often missed and more often inadequately treated; three applications of gentian violet in 1% solution at twelve-hour intervals were effective in our cases, even with the doctor's child referred to above; the whole of the mucous membrane of the cheeks, particularly between lips and gums and tongue, is painted. Thrush is a serious threat to life in premature and sickly babies if allowed to progress to œsophagitis and enteritis or generalized monilial infection, and as the organism is being constantly reintroduced to maternity hospitals by infected mothers I feel that it is a tribute to our nursing staff that no monilia was recovered from bedding or dust, that the incidence was low and that there were no "thrush buttocks".

[1] This child was a normal, healthy, breast-fed infant born by normal delivery, but it cut a tooth as soon as the thrush subsided, revealing red, swollen gums. This may have been the local factor influencing the development of thrush.

Umbilical Infection.

I had great difficulty in assessing umbilical infection, so that the figures are not clinically or pathologically accurate. For the purposes of the investigation an "infected cord" was any offensive cord, any umbilicus with local inflammatory reaction, or any case in which the baby's general condition was suggestive of infection in which a pathogen was recovered from the umbilicus and in which there was no other apparent cause of infection (one case, see below). The pathological investigation we carried out (Coventry and Isbister, 1951) revealed that 54% of the three-day to four-day old babies were carrying coagulase-positive staphylococci on the umbilicus, and that by the sixth day 58% were yielding positive culture results. This made it very important to correlate the clinical and pathological findings.

There was only one patient with pyrexia, wasting, jaundice and apparent severe infection; in this case hæmolytic *Staphylococcus aureus* was grown from material taken from the umbilicus and the child responded to chemotherapy. In accordance with Professor Capon's classification (Paterson and Moncrieff, 1947) this was probably the only case of umbilical phlebitis and arteritis. There were no cases of gangrenous omphalitis. All the rest probably fit into the category of purulent omphalitis with or without umbilical granulomata.

Most of the offensive, late-separating wet cords yielded cultures of *Bacillus coli communis, Proteus vulgaris* or *Bacillus pyocyaneus*; they produced little if any general disturbance in the baby and responded well to three-hourly dressings with spirit. No chemotherapy was given unless there was general reaction. Many cases were observed to be associated with small granulomata that persisted after separation of the cord. As Professor Capon notes, some of the children affected did leave hospital with small umbilical herniæ or protuberant umbilici. When hæmolytic *Staphylococcus aureus* was considered to be the infecting organism, there appeared to be less local than general reaction, the cord was not so offensive or wet, and in most cases it did not separate later than usual, but the babies were jaundiced and lethargic.

Since realizing the frequent occurrence of *Staphylococcus pyogenes*, we have particularly observed all jaundiced babies, never accepting the dangerously complacent diagnosis of "physiological jaundice" until other explanations have been exhausted. At the time of writing there have been four cases in the last month in which the baby was jaundiced on the fourth day, lethargic and not obviously ill; two of the babies were afebrile but difficult to feed and generally regarded by the nursery sister as not progressing satisfactorily; from three *Streptococcus hæmolyticus* was grown and from the fourth hæmolytic *Staphylococcus aureus*. Their general condition responded immediately to chemotherapy, and the jaundice also decreased rapidly, but took several days to disappear. It appears to me that hepatitis and septicæmia may be commoner lesions in babies than the more often quoted pylephlebitis and liver abscesses, a conclusion supported by Morison in Belfast (1944).

Respiratory Infection.

There were only two cases of purulent rhinitis (compare Abramson, 1949). The diagnosis of pneumonia was rarely made as most cases of illness associated with rapid respiratory rate, cyanosis and pyrexia occurred on the first two days of life and were regarded, after consideration of labour *et cetera*, as being primarily cerebral in origin with atelectasis. It is obvious that in such cases the children would easily become infected and they received chemotherapy. When chest X-ray examination was carried out it was considered that one could not be dogmatic about whether a shadow was an area of collapse or pneumonia in a child at that age. Post-mortem examinations on babies who died did appear to support the primarily cerebral nature of these cases. When there had been inhalation of vomitus, food or potentially infected material at birth, or when there had been ruptured membranes for more than twenty-four hours, pneumonia was considered the more likely diagnosis, but the recovery of *Bacillus coli* or *Staphylococcus pyogenes* from nose or throat after the second day probably had little diagnostic value. It is probable that I have under-estimated respiratory infection, and I did not observe any association with conjunctivitis (Campbell, 1951).

During the period under consideration no cases of urinary infection were detected nor any of *otitis media*; both were borne in mind but perhaps not searched for as diligently as was desirable.

There was no case of meningitis and only one of gastro-enteritis (compare Field, McCarthy and Wyllie, 1943). Both conditions were diligently sought and any suspicious green motion meant isolation until a diagnosis was made.

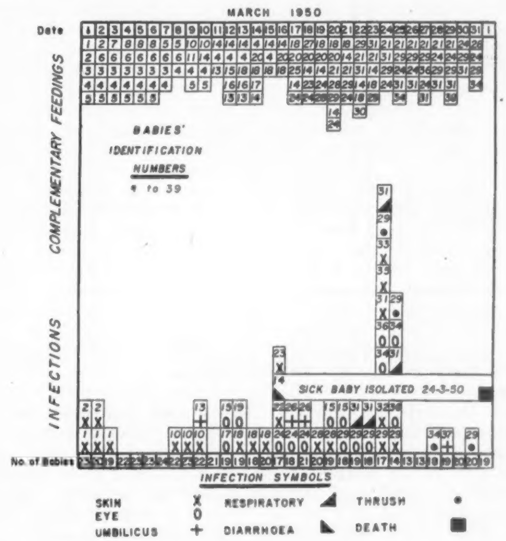

FIGURE III.

Illustrates the method of charting daily the infections of the newborn and complementary feedings in one nursery for the month of March, 1950, when the maximum number of infections was recorded. In the original charts colours were used to represent the infections instead of symbols. The total number of babies in the nursery each day was also charted, and it can be seen that the small epidemic of staphylococcal infections was associated with a busy month, and the nursing of a sick baby (number 14) with cerebral damage, in the nursery.

METHODS OF COLLECTION OF INFORMATION.

Spence charts (Figure III) were kept for December, 1949, and March, May and June, 1950; the infections occurring in January, February and April were recorded by the method of daily personal examination of babies reported as having any abnormal finding, and the results were examined. During the period of the combined pathological and clinical examination, every baby, normal or reported abnormal, was fully examined, and during the first three months of the period an attempt was made to examine every baby in order to get a clear idea of the range of normal as well as what was passed as normal by the nursing staff. Therefore, I feel sure that the figures for minimal infections of thrush, conjunctivitis and pustular rashes are reasonably accurate, but it is very difficult to determine what is a mildly infected cord.

As noted by Professor Spence and his co-workers (1946), it did appear that infections were greatest when the wards were fullest and that the infections were commonest among the babies receiving complementary feeding. However, in our case it was not policy to complement feeding unnecessarily. This investigation was being carried out secondarily to a lactation investigation, and the primary work under consideration was the establishment of lactation, so that the mother's breasts were always emptied and the baby was not always placated. We did test feed, but in a

TABLE I.
Distribution of Infections.

Month.	Nursery.	"Sticky Eye."	Pustules.	Thrush.	Umbilical Infection.	Respiratory Infection.	Paronychia.	Number of Infected Babies.	Total Births.	Percentage of Infected Babies.
February ..	1 F	1	4	1	3	—	1	8		
	2 F	—	2	—	—	1	—	3 (26)	161	16·2
	An	7	2	—	4	—	—	12		
	P	—	1	1	—	1	—	3		
March ..	1 F	6	10	1	3	2	1	19		
	2 F	—	2	—	1	—	—	3 (39)	164	23·8
	An	7	7	—	1	—	—	15		
	P	—	1	—	1	1	—	2		
April ..	1 F	1	3	—	1	—	—	6		
	2 F	4	8	1	1	—	—	11 (33)	147	22·4
	An	3	8	1	1	—	—	12		
	P	1	2	3	1	—	—	4		
May	1 F	2	3	1	2	1	—	6		
	2 F	4	2	1	1	—	—	7 (32)	180	17·8
	An	8	3	—	5	—	2	17		
	P	1	—	—	—	—	—	2		
June	1 F	4	2	2	7	1	—	13		
	2 F	3	2	—	—	—	—	5 (32)	150	21·3
	An	6	5	—	3	—	—	13		
	P	1	—	—	—	—	—	1		
December ..	1 F	9	1	—	—	—	—	10		
	2 F	4	2	3	—	—	—	7 (30)	138	21·7
	An	7	3	—	—	—	—	10		
	P	—	—	1	—	3	—	3		
Total ..	.. ..	79	73	17	35	10	4	192	940	20·4

Total infections = 222. Total infected infants = 192. Neonatal deaths (none due to infection) = 21. 1 F = First floor. 2 F = Second floor. An = Annexe nursery. P = Premature nursery.

busy obstetric hospital with our nurses working a forty-hour week and babies in nurseries it is very difficult to know what is happening to lactation any other way, particularly as the mother is usually discharged from hospital before the tenth day but not before the seventh. Boiled water was given as a routine, and it is difficult to see how we could avoid this with our oppressive hot January and February weather. The careful asepsis of these procedures is proved by the low incidence of thrush.

I therefore decided to attempt to determine whether there were not other factors involved that made these babies require complementary feeds as well as contract infections. Unfortunately, all the information concerning labour, ante-natal history, puerperium and feeding difficulties, and neo-natal disease other than infections, had to be collected in retrospect. The sources of information were the infant report book, the labour ward record, the maternal report book, the nursery books, the maternal history (official hospital records) and the baby's history (official hospital records). These were examined in detail and the results tabulated. It is worthy of note that the nursing records proved more detailed and considerably more accurate than the medical records, and that only about one-third of the infections were recorded on the official histories, whereas the sister's report book agreed closely with the results of my examinations. I note this particularly, as anyone attempting to assess the infection rate in a maternity hospital may be completely misled by the histories. In fact, it may be stated that unless the resident medical officer in charge of nurseries (a non-existent person in many hospitals) does a daily round and records the infections present as he does that round, the results will be totally inaccurate. This is very apparent when one compares the histories of a conscientious interested resident medical officer with those of the resident medical officer who obviously writes his histories from memory after the discharge of the patient from hospital.

INCIDENCE OF INFECTION.

I have attempted to record the infections occurring in The Royal North Shore Hospital nurseries using the same criteria of diagnosis as those used by Dr. J. D. Roscoe in Cambridge (1949), Dr. Mary Taylor at Queen Charlotte's Maternity Hospital (1949), Professor J. C. Spence and his assistants in Newcastle (1946, 1949), Dr. M. J. L. Frazer in Belfast (1948), and Dr. J. L. Henderson in Edinburgh (1943). However, I have comparatively high figures for umbilical sepsis (19% of the total); these are mainly responsible for making our figures appear higher than those of Dr. Roscoe, and it seems that my criteria of diagnosis were not the same. I do feel sure, particularly as we conducted a pathological investigation at the same time, that the cords concerned were infected and potentially dangerous. I have also recorded paronychia, not recorded in some other figures, and I have taken the term "green stool" as too vague and decided that there was only one green stool that might possibly have been infective in our series. I stress the fact that minimal infections were recorded and that there were no deaths. There are four nurseries: two twenty-cot nurseries in the obstetric block, a premature baby nursery of seven cots, and an annexe nursery in a temporary building to which about half the mothers and babies are transferred on their fourth day or later provided the mother is well and able to get up.

Table I shows that the incidence of infection of all types was 20%, as compared with that reported by Dr. J. D. Roscoe of Cambridge (10%), Dr. J. L. Henderson in Edinburgh (29%), Dr. B. Corner in Bristol (27%), and Dr. M. J. L. Frazer in Belfast (under 3%); the last-mentioned excellent results were achieved mainly by a lowering of the incidence of eye infections.

The figures for January were omitted from Table I as they were not quite complete, but the infections studied in detail are 185 consecutive cases from January 15 to June 30.

Lest it be said that these figures appear to show a high incidence of mild infections, I hasten to add that I visited all the baby health centres in neighbouring districts while carrying out a lactation investigation and asked casually what the infection rate among their newborn was like. I nearly always received the answer: "There are hardly ever any pustules and never any thrush in the Royal North Shore Hospital babies—that is partly why the hospital has such a good reputation", and the hospital reputation appeared very high. I attempted to gauge this a little more accurately by examining the admissions to Carpenter House and studying the patients with infections admitted from different hospitals with the following results (Table

II). At The Royal North Shore Hospital the baby's "free of infection" certificate was issued by the resident medical officer, and I made no attempt to examine babies before they left for Carpenter House, so I am sure that The Royal North Shore Hospital figures were not influenced by me.

Factors Influencing Infection.

In attempting to assess the factors influencing infection we must consider the elementary points, namely: (i) the dose of the organism received; (ii) the virulence of the organism; (iii) the resistance of the patient to the particular organism considered.

Dose of Organism.

Many investigations have now been carried out in maternity hospitals that adequately demonstrate how frequently coagulase-positive hæmolytic *Staphylococcus pyogenes* is found in the noses and throats of mothers, nurses, and babies (Roscoe, 1949; Rountree, 1950). In our own small investigation we took nasal and umbilical swabbings daily from 45 babies during their stay in hospital (a total of 175 swabs) and found that 42·9% of nasal swabbings and 49·7% of umbilical swabbings yielded growths of coagulase-positive *Staphylococcus pyogenes*. We took swabbings from 12 nurses and wardsmaids, and from powder, oil, swabs, spirit, *et cetera*, but we grew hæmolytic *Staphylococcus pyogenes* only from swabbings from one nurse and from a hand towel (Coventry and Isbister, 1951). There seems little doubt that pathological organisms are freely available to the baby and that many are penicillin-resistant. It seems, too, that cross-infection from baby to baby by hands of nurses is the most likely method of spread. In support of this it was our experience on several occasions that our infections increased in number when the nursery was full and when we were nursing a sick baby in the nursery for a longer period than the usual stay (note the March chart). It would obviously reduce the dose to observe more carefully the rules of asepsis, and the fact that we had very little hæmolytic *Staphylococcus pyogenes* in our premature nursery, despite the notorious lack of resistance of premature babies to infection, indicates that our more rigid asepsis there was successful. The use of paper hand towels, as advocated by Dr. Mary Crosse (1949), would seem desirable as a way of breaking the chain of cross-infection.

TABLE II.
Carpenter House Admissions.
January to June.

Hospitals.[1]	Number Admitted.[2]	Eye.	Pustules.	Thrush.
1	10	—	—	1
2	9	1	1	1
3 (Royal North Shore Hospital)	21	1	2	1
4	11	2	3	1
5	4	1	1	—
6	22	3	5	12
7	6	—	—	—
8	7	1	3	—
9 (Other hospitals)	18	1	1	2

[1] 1–8 represent maternity departments of hospitals, Royal North Shore Hospital being number 3.

[2] No sick baby was admitted. There were no infections that did not respond rapidly to treatment. Two babies were sent back to hospital after admission: (i) a baby with pyloric stenosis, (ii) a jaundiced baby with green stools, who ultimately turned out to have umbilical sepsis. There were a few cases of jaundice and doubtful conditions of the umbilicus, but as figures were collected from records and I did not personally examine them all, I have not included them.

Virulence of Organism.

We attempted only to determine clinical infections and to ascertain, where possible, if the organism was a pathogen, and in the case of *Staphylococcus pyogenes* whether it was hæmolytic and coagulase-positive. Determination of penicillin and streptomycin resistance was requested only if the child had had a course of penicillin already and we knew nothing of strain details. I was more impressed with the increase in staphylococcal infections in full nurseries during hot weather, particularly when we had one sick baby, than I was with the possibility of epidemic strains, but the work of Allison and Hobbs in Cardiff (1947) seems to show fairly conclusively that epidemic strains occur. Our own epidemic was in March, when we rapidly closed the nursery for three days, scrubbed, aired, and generally washed it with antiseptic, and had no further trouble. The sister in charge tells me that the only previous such event in her memory, which goes back some fifteen years, was traced to a shortage of washers, and the restoration of individual sterile washers for each baby eliminated infection.

I think it significant that very few of our patients failed to respond rapidly to penicillin and local treatment, though we did recover penicillin-resistant strains from two babies who had previously had courses. It suggested to me, possibly quite erroneously, that though our endemic strains were penicillin-resistant, new penicillin-sensitive organisms were constantly being introduced, possibly by the mothers, as appeared to occur in the case of thrush; the results of examination of vaginal swabbings from the mothers might have proved interesting.

I am inclined to doubt very seriously the statement in the Ministry of Health Neo-natal Mortality and Morbidity Report (1949) that infected breast milk may infect the baby. I was carrying out a mastitis investigation concurrently with this investigation, and in 13 cases the baby had an infection, clinically staphylococcal, before the mother's infection. In the cases in which the day of infection was noted (nine) eight occurred less than five days after the babies' infection (note the similarity to the circumstances shown on the pustules chart). In each case in which breast milk was submitted to culture, the organism was hæmolytic *Staphylococcus aureus* (in none of my series was any other pathogen grown, and the results were 100% positive when culture was carried out). There seems little doubt that the baby passed the virulent organism acquired in the nursery to the mother, in these cases at least.

Resistance of the Patient.

It is extremely difficult to assess the resistance of the patient, but it seemed reasonable to collect all available information about the mother's labour and the health of mother and baby while in hospital to see if any relevant facts arose. I have been unable to learn what constitutes local or general resistance to *Staphylococcus pyogenes*, and this makes the problem even more difficult.

The factors tabulated were labour of mother, parity of mother, maternal health (particularly presence of any infection), feeding of infant, birth weight, and condition of baby at birth and afterwards. One hundred and eighty-five consecutive infected babies were investigated.

Parity and Labour.

The Report on Neo-natal Mortality and Morbidity states that "the stillbirth and neo-natal mortality rates are relatively high in primigravidæ, at the lowest in second pregnancy, then rise in each succeeding pregnancy until in the fifth it is as high as the first, and thereafter it rises still further with each succeeding pregnancy". I think it would be generally conceded that the baby usually goes through a more severe ordeal if its mother is a *primipara*, and that abnormal presentations and precipitate deliveries are likely to distress the baby. Unfortunately the mothers in my series are classed as *multiparæ* if they have ever been pregnant, so I do not know how many are virtual *primiparæ* as far as labour is concerned; I also cannot say how many had passed into the danger period after the fifth pregnancy.

In my series the following figures were obtained:

Total number of births between July 1, 1949, and June 30, 1950, 1701. Births by instrumental delivery, 154 (9%). Births by Cæsarean section, 38 (2·2%).

Babies with infection investigated, 185. Infected babies born by instrumental delivery, 26 (14%); expected number, 17. Infected babies born by Cæsarean section, eight (4·3%); expected number, four.

It is worthy of note here that the majority of women having surgical or instrumental intervention receive chemotherapy, and the majority of babies with trauma or asphyxia would have received chemotherapy prophylactically and would not, as actually occurred, appear in the series of those with infections; I think it is likely that without the prophylactic use of penicillin our infection rate and our percentage of abnormal deliveries with infections would have been higher.

Total number of births between January 1 and June 30, 1950, 937. Births from *multiparæ*, 615. Births from *primiparæ*, 322.

Babies with infections investigated, 185. Infected babies born of *multiparæ*, 108. Infected babies born of *primiparæ*, 77; expected number, 65.

An analysis of the labours of the 108 *multiparæ* whose babies had infections while in hospital revealed that 54 had abnormal labour—that is, 50%.

The term abnormal labour was by no means what Dr. Grantly Dick Read would mean. I took the term as including cases reported as such by the labour ward sister and added to these the cases of patients with toxæmia who had required induction of labour, and cases in which the baby required artificial respiration. Only severe post-partum hæmorrhages were included as abnormal, and perineal tears were not classed as abnormal. There were many very short labours in this series of normal cases, but I felt that for the baby these might have been precipitate, and we have found repeatedly that a precipitate delivery will often distress the child more than a long labour with an instrumental delivery. In the entire series of *multiparæ* and *primiparæ* 80 mothers were recorded as having normal labours and 108 abnormal. I have not analysed the normal babies without infections to see the ratio of normal to abnormal labours, but it appears that when there are factors in the labour that may have made it more difficult for the baby to accommodate itself to its new environment, that baby is more likely to become infected.

The following is an analysis of the remaining 54 cases in which the baby of a *multipara* became infected after a normal delivery: 10 mothers had puerperal infection, either gynæcological or nasopharyngeal; 10 babies were artificially fed or lactation was rapidly failing; three babies were ill for other reasons than infection; two mothers had ante-partum ill health of a severe nature (one had severe anæmia and the other syphilis which was treated). Among the remaining 29 cases there were several in which I felt that overwhelming dose appeared to negate the lowered resistance factor, as five of the babies were in the nursery that we closed because of infection and some of the others were among those who developed eye infections after going to the dustier atmosphere of the annexe (see Figure I).

Also, these histories were all regrettably collected in retrospect, and since making these observations we have noted more carefully the labour ward story while it was fresh in the mind of the staff; we have found that the labour often proved a greater strain to the baby than the record indicated. Signs of fœtal distress, particularly meconium-stained liquor and yellow vernix, were not always written up, and the fœtal heart variations were not noted by me in recording the figures. I am sure a fuller recording of the babies' condition on delivery and more searching examination of the records would have revealed more shock and temporary "lowered resistance" among the babies.

Examination of the Carpenter House series shows that there were 39 *multiparæ* admitted in the six-month period and 11 infections among their babies. The infection was in the form of pustules in six cases, sticky eyes in two, and thrush in three. Delivery was instrumental in two cases, by Cæsarean section in three, and premature in three. Nine of the eleven infections occurred in association with the eight abnormal labours, and of the other two one occurred in a baby weighing 11 pounds at birth. The figures are small but tend to confirm the Royal North Shore Hospital figures.

Feeding.

Investigation of the feeding of the babies in the 185 cases investigated showed that 17 were artificially fed on discharge from hospital, 41 were partially breast-fed, 111 were fully breast-fed, and in 16 cases there was no record.

These figures indicate that at least 58 of the 185 babies —that is, 31·3%—were still presenting feeding problems on discharge from hospital and required complement. My records were not sufficiently accurate to indicate how many of the babies discharged from hospital fully breast-fed had required complement in hospital, but during the months that I did keep accurate Spence charts of infection and feeding together it was apparent that infections occurred more frequently among those receiving complement or being artificially fed.

I have no really satisfactory comparison, but it may be helpful to state that I was carrying out a lactation investigation at the same time among *primiparæ*, and of 139 consecutive cases occurring in three months of the six-month period under study, the baby was at the time of discharge from hospital being artificially fed in six, partially breast-fed in 30 and fully breast-fed in 103. Thirty-six of the 139 babies (records were complete) were still presenting feeding problems on discharge from hospital (that is, 26%). I think that this percentage would have been lower if the *multiparæ* had been included, as it is my impression that we discharge from hospital a higher percentage of *multiparæ* fully breast-feeding their infants than *primiparæ*, and Waller has satisfactorily demonstrated that *multiparæ* establish full lactation securely before *primiparæ* (Waller, 1947).

As my series covers only two weeks of the neo-natal period, and as the majority of infections occurred in the first week, I find it hard to believe that complementary feeding was causative and regard it rather as associated. It may possibly have tended to aggravate the lowered resistance of the child, and the high incidence of thrush amongst the artificially fed and its low incidence among the breast-fed suggest, as has been suggested before, that the altered pH of the milk may be a predisposing factor.

I do not think that there is any doubt that it is the babies receiving complement and having feeding difficulties that develop infections, but the nature of the mother's labour and other predisposing factors may cause both the feeding difficulty and the state of lowered resistance to infection in the baby; for example, it was not unusual in our series to observe both delayed and inefficient lactation in a primiparous woman after a difficult labour and a tired baby who was late in going to the breast and sucked poorly when it did go, with resultant feeding problems.

Prematurity.

I can merely make a few observations but cannot attempt any assertions on the predisposing effect of prematurity, as I completely accept the generally believed statement that premature babies are more liable to infection than full-term babies, and therefore we did our best to prevent infection; we nursed these babies in a separate air-conditioned nursery with strict asepsis and used prophylactic penicillin therapy more frequently than in the general nurseries. Of the eight infections that actually occurred in the premature ward during this period there were two infected cords (offensive and presumably due to *Bacillus coli communis*), two infected eyes, two pustules, and two cases of thrush. It was apparent from the charts that there was a much lower incidence of staphylococcal infection in the premature ward than in the others, owing presumably to greater asepsis. The two cases of pustules were in babies born in private hospitals and admitted to our hospital on their second day, a practice no longer permitted, as our premature ward is not big enough to allow satisfactory isolation of such babies. Three further cases of staphylococcal infection occurred among the premature babies after their transfer from the premature ward to the general nursery.

Another group that proved interesting and instructive were the babies above five and a half pounds in weight

but born early by the mothers' dates—for example, the six and a half pound baby of a mother who had previously had an eight pound baby and said that she was not due for a month. Unfortunately, I did not record the estimated date of confinement in these cases, but I had at least 13 such cases among 99 in the two main block nurseries. The babies concerned acted as premature babies and gave us more trouble than some of the babies under five and a half pounds; they were facing the harder existence of the general nursery and developed more infections. They are, in fact, a very interesting group that need special care, but they suffer because of the international definition.

Effect of Local Factors.

Unfortunately, it was not noted in the reports as a routine whether a baby was being bathed or oiled, and though it is supposed to be noted on the hospital history whether or not the skin is dry, this was not always done; so I have no figures on the incidence of dry or peeling skin. The nursing staff are firmly convinced that babies with this condition and those with prickly heat are much more likely to get pustular rashes. This needs statistical proof, but I feel sure that if it was investigated we should find that babies with little or no vernix, dry and peeling skin, or prickly heat, are more liable to pustular rashes. We reduced the number of baths, used an animal fat ("Lanoline") instead of vegetable oils, and only "topped and tailed" the dry-skinned babies, with the most gratifying results until February and March, 1951, at the time of writing this article, when with hot humid days and overcrowded nurseries there was a sudden increase in the incidence of prickly heat and a steep rise in the incidence of pustular rashes.

In the annexe "sticky eye" chart (Figure I) I think that there is sufficient evidence to incriminate dust as a local irritant predisposing to "sticky eye". This observation has already been made by Dr. J. D. Roscoe (1949).

The handling of babies was reduced to a minimum, nurses changed babies in the cots, and the number of baths was reduced (first and fourth day, then daily if the skin and general condition were normal). Babies who had had instrumental or Cæsarean deliveries, or had been in any way affected by delivery, were cot-fed for the first forty-eight hours.

SUMMARY.

1. An attempt has been made to record all the infections, even the most trivial, occurring in four nurseries attached to a maternity department over a seven-month period, the methods of Professor J. C. Spence being used.

2. The infections include the mildest as well as severe forms, and it is stressed that such infections are frequently unrecognized by medical and nursing staff caring for the newborn, as is evidenced by the number of infected babies admitted to a mothercraft home accompanied by a certificate stating absence of infection.

3. The common infections of the newborn are discussed in detail and described as they occurred in this series.

4. The histories of 185 babies with infections (consecutive cases) were examined in an effort to determine any predisposing factors. Particular note was made of parity of mother, mother's labour and ante-partum and post-partum health, baby's birth weight and feeding, and the day of onset of infection. It is concluded that the same factors that tend to cause the baby "to fail temporarily to establish the mechanism of independent existence" (Spence) are associated with the occurrence of infections in the newborn period, particularly the first week of life. It is suggested that these factors may cause lowered resistance of the baby to infection.

5. It is suggested that the great preponderance of pustular rashes on the fourth and fifth days of life may cast some light on the incubation period of staphylococci in skin infections, as it seems certain that exposure to infection could have occurred only during or after delivery.

6. The prevalence and potential danger of umbilical infection are emphasized.

CONCLUSION.

In Australian cities and even in most country areas obstetrics is now carried out almost entirely in hospitals; the maternal and neo-natal death rate has fallen, but, as in other parts of the world, more emphasis is now being placed on the infections that occur in the nurseries of the maternity hospitals. So great is this emphasis that we often hear thinking, responsible people say that women should have their babies at home, and the idea is spreading to mothers, many of whom feel that they have no choice and that hospital is a necessary evil; the result is that they want to leave as soon as possible, so delaying the establishment of lactation and their return to normal health. I shudder to think of the results of the forty-eight-hour stay in hospital that exposes the baby to the hospital staphylococcus and sends the mother home to struggle with her lactation, her puerperal state and the household duties. My series of cases includes the minimal infections, and though their incidence appears to be high, there were no deaths due to infection and very little morbidity. The incidence can undoubtedly be lowered by more meticulous asepsis and smaller nurseries or possibly, as Professor Spence advocates, by abolishing nurseries and by nursing mother and baby together. This is not practicable in many of our hospitals with large wards without considerable complication of nursing, but it should be possible in one-bed and two-bed wards.

It seems to me from the cases studied that there is a much greater incidence of infection amongst the babies of mothers with complicated labours, and it would be a serious retrograde step to encourage women to think that *primiparæ* and the majority of *multiparæ* could have their babies other than with the best facilities, in the hope of reducing infection. We might again be losing babies who now get only a mild infection, rapidly cured if diagnosed early. I feel that we are a generation that is forgetting the horror of confinement in a feather bed (or would it be an inner-spring mattress?) and of an instrumental delivery with newspaper drapes after a kitchen-sink scrub-up.

I wish to emphasize that the infection rates can be reduced, but only if nursing and medical staff are alert to the risks of infection and are able to diagnose the early manifestations of infections, particularly staphylococcal—a state of affairs that certainly does not appear to exist in the general community at present. The establishment of a nursery for babies who have had a stormy entry into this world, where they could be nursed under premature nursery conditions for forty-eight hours, would, I think, reduce the infection rate.

The distribution of "sticky eyes" strongly suggests that dust is an aggravating factor, and all efforts to lay the dust by oiling or washing floors rather than sweeping dry floors will help to reduce infection.

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A BACTERIOLOGICAL AND CLINICAL STUDY OF INFECTION IN NEWBORN BABIES IN A MATERNITY HOSPITAL NURSERY.

By KEITH J. COVENTRY, M.B., B.S.,
Pathology Registrar, The Royal North Shore Hospital of Sydney,

AND

CLAIR ISBISTER, M.R.A.C.P., D.C.H.,
Honorary Assistant Pædiatrician to the Obstetric Department, The Royal North Shore Hospital of Sydney, Sydney.

As the neonatal death rate falls slowly with improved hospital facilities and better care of the premature baby, more emphasis has been placed on infections.

There seems to be little doubt that because more women are having their babies in hospital, the babies are exposed to a greater risk of cross infection with pathogenic organisms (Cunliffe, 1949). A high proportion of these are *Staphylococcus pyogenes* (for our use of the term *Staphylococcus pyogenes* see later under "Bacteriological Methods"). The fact that many of these organisms are penicillin and streptomycin resistant has caused grave concern.

Most of the articles hitherto published have discussed in detail either the bacteriology of the definite infections reported in the nurseries or the bacteriology of the normal nursery. Although a number of investigations have been carried out upon the nasal carrier rate of *Staphylococcus pyogenes* in babies (Cunliffe, 1949; Martyn, 1949; Rountree and Barbour, 1950), little interest has centred on the bacteriology of the umbilical cord. In our experience several cases occurred in which a pathogenic organism was grown from umbilical swabbings, with the result that discussion arose on whether a pathogen was likely to be grown from any umbilicus or whether the baby's clinical picture was due to umbilical infection. It was decided to attempt to gauge the significance of this finding by taking umbilical swabbings from a larger series of babies, sick and well. It seemed that it might be valuable to correlate the findings from these with those from nasal swabbings taken from the same infants.

There has already been much valuable detailed work (Cunliffe, 1949; Martyn, 1949; Rountree and Barbour, 1950) on the bacteriology of the nursery and its infections. However, it is felt that in some surveys investigations were confined to well-developed infections. In this series it was the particular aim to discover the very earliest clinical signs of infection.

This investigation was carried out at the Royal North Shore Hospital of Sydney during June, 1950. It comprised clinical and bacteriological examination of all the babies in a twenty-bed nursery, over a period of ten days. At the time there appeared to be an average amount of clinical infection among the babies.

Methods Used in the Nursery.

Every baby in the nursery was fully examined every day by the pædiatrician with the help of the resident medical officer; special note was made of its skin, eyes, nails, umbilicus, nose and mouth, and of the clinical condition otherwise—for example, jaundice, respiratory distress *et cetera*. A serious effort was made to determine clinically the existence of even the mildest infection.

The history of every baby and of its mother was recorded as regards health of the mother, labour, condition at birth, feeding and neonatal incidents.

Nasal and umbilical swabbings were taken daily, between 10 and 11 a.m., from every baby in the nursery. Before use swabs were moistened with nutrient broth, of which a fresh tube was used for every three babies, the used tubes being subsequently submitted to culture. The broth was sterile after use on every occasion. The nasal swabs were inserted one or two centimetres into one nostril, and the umbilical swabbings were taken from the base of the cord.

Any babies with clinical evidence of infection had swabbings taken from the infected area—for example, eye or pustule.

Swabbings were taken from the nose and fauces of each medical officer, nurse, and wardsmaid working in the nursery.

A few swabbings were taken from substances within the nursery, namely, dust, blankets, umbilical dressings, swabs, oil, dressing powder, and a hand towel.

Bacteriological Methods.

All material was inoculated onto blood agar plates, within half an hour to two hours of collection, and examined after twenty to twenty-four hours' incubation at 37° C.

From each culture containing staphylococci a typical colony was emulsified in a tube of nutrient broth. After six hours' incubation, this broth was used to inoculate media for coagulase and sensitivity estimation.

Coagulase activity was estimated by adding five drops of the broth culture to 0·5 millilitre of a 1:10 dilution of human plasma. This was examined after one hour, and after incubation for twenty-four hours at 37° C.

All coagulase-positive strains were classed as *Staphylococcus pyogenes* (following Cowan and Shaw, 1949, and Rountree and Thomson, 1950), irrespective of their pro-

duction of pigment or hæmolysis. All coagulase-negative strains were classed as *Staphylococcus saprophyticus.*

Sensitivity tests were carried out by streaking a loopful of the six-hour broth culture on nutrient agar plates, containing (*a*) 10 units of penicillin and (*b*) 10 microgrammes of streptomycin per millilitre of medium. Control cultures of sensitive Oxford strain staphylococcus were included on each plate. Each culture was also inoculated on an agar plate not containing an antibiotic.

Strains were classed as resistant if any colonies were visible on the plates containing antibiotic after twenty-four hours' incubation at 37° C.

Cultures were not tested for penicillinase production or phage type.

Results of Investigation.

Examination of nasal and umbilical cultures showed a rapid rise, with increasing age of the babies, in the percentage carrying *Staphylococcus pyogenes.* This is shown in Table I.

During the period under consideration forty-four babies occupied the nursery. Some were over seven days old when the investigation commenced, and some only one day old when it ended.

Colonies of *Staphylococcus pyogenes* were grown from the noses of 30 babies at some period during their stay, and from the umbilici of 31.

TABLE I.
Results of Examination of Swabbings from Healthy Babies.

Age of Babies. (Days.)	Number of Swabbings Taken.	Number of Babies Carrying *Staphylococcus pyogenes.*	Number of Babies Carrying *Staphylococcus pyogenes* in Umbilicus.
0 to 2 ..	31	8	6
3 to 4 ..	50	17	27
5 to 6 ..	36	18	21
7 to 14 ..	58	32	33

Both the nasal and umbilical swabbings were sterile from seven babies, all of whom were less than two days old; of seven babies whose nasal swabbings only were sterile, two were premature babies over a month old, three were three days old, and two were one day old; of six babies whose umbilical swabbings only were sterile, one was over a month old, two were three days old, and three were less than two days old.

Eight babies developed clinical infections during the investigation period (see Table II).

In Cases III and IV *Staphylococcus pyogenes* was grown from nasal swabbings. In Case V the patient was a day-old baby and it is probable that the swabbing was taken after local penicillin treatment had been given.

Seven babies were jaundiced. From the umbilical swabbings of five of these *Staphylococcus pyogenes* was grown, and from one *Bacillus coli* was grown; the seventh, who developed jaundice two days after the investigation finished, had had a sticky eye on its first day of life.

Results of Sensitivity Estimations.

A high proportion of the strains of *Staphylococcus pyogenes* were found to be resistant to penicillin and to streptomycin. The findings were as follows:

Total number of cases in which *Staphylococcus pyogenes* was isolated from noses and umbilici of healthy babies	171
Number of strains penicillin resistant ..	104 (61%)
Number of strains streptomycin resistant	92 (54%)
Number of strains resistant to penicillin and to streptomycin	64 (37%)

Results of Investigations on Attendants and Fomites.

From only one of the twelve members of the staff was *Staphylococcus pyogenes* grown. This organism was sensitive to penicillin but resistant to streptomycin. *Staphylococcus pyogenes,* resistant to both penicillin and streptomycin, was grown from a blanket on one of the nursery cots. A profuse growth of *Staphylococcus pyogenes,* which was sensitive to penicillin and to streptomycin, was grown from a hand towel. From about a dozen swabbings taken from other articles in the nursery no organism likely to be pathogenic was demonstrated.

Discussion.

Other investigators (Cunliffe, 1949; Martyn, 1949; Rountree and Barbour, 1950) have pointed out the rapidity with which the nasal mucosa of a baby in a hospital environment becomes populated with *Staphylococcus pyogenes.* Thus Martyn (1949) isolated this organism from 62% of 130 babies aged nought to seven days; and Cunliffe's figures (1949) showed a progressive increase in the nasal carrier rate from 9% on the first day of life to 96% in a group aged seven to fourteen days.

TABLE II.
Clinical Infections.

Case Number.	Area.	Result of Culture.	Sensitivity to.	
			Penicillin.	Streptomycin.
I	Pustular rash.	*Staphylococcus pyogenes.*	Resistant.	Resistant.
II	Pustular rash.	*Staphylococcus pyogenes.*	Resistant.	Sensitive.
III	Pustular rash.	*Staphylococcus saprophyticus.*	Sensitive.	Sensitive.
IV	Conjunctiva.	Sterile.	—	—
V	Conjunctiva.	Sterile.	—	—
VI	Umbilicus.	*Bacillus coli communis.*	—	—
VII	Umbilicus.	*Bacillus coli communis.*	—	—
VIII	Umbilicus.	*Staphylococcus pyogenes.*	Resistant.	Sensitive.

Our series is too small to enable any definite conclusions to be drawn, and pressure of routine bacteriological work made it impossible to enlarge the series. Nevertheless, we believe that, as our findings from nasal swabbings agree substantially with those of other investigators, we are justified in drawing attention to the results from the umbilical swabbings.

In 30 of the 44 babies we were able to demonstrate the presence of *Staphylococcus pyogenes* in the nose, but, after all, this is a region where organisms are filtered from the air and are to be expected.

Despite a strictly supervised aseptic routine for care of the umbilical wound, it was possible to recover *Staphylococcus pyogenes* from the umbilicus of 31 of the 44 babies. This is a more disturbing finding, particularly as the incidence of positive results from swabbings was at its highest among the babies three to six days old, when the cord was separating.

Here an open wound, communicating with the liver and large blood vessels, has been exposed to a virulent infection, and local resistance would not be expected to be as high as in the naso-pharynx.

It became immediately apparent that a positive result from an umbilical swabbing merely indicated exposure to infection and told us nothing of the baby's health unless correlated with the clinical picture.

Well-marked umbilical infection leading to liver abscesses and septicæmia has long been recognized as a cause of neonatal mortality (Trousseau, 1869). However, the literature does not appear to yield a clear clinical picture of mild or early umbilical sepsis. A statement by Morison (1944), of Belfast, in which are reported the pathological findings after nine deaths from umbilical sepsis, is worth quoting:

> A review of the clinical histories in cases with hepatitis, showed that in all cases the disease process was well established by the sixth day. . . . In the present cases the lesion at the umbilicus was often slight. At

the time of death, there was in most of them no evidence of a septic thrombophlebitis or any active infection proximal to the granulation tissue blocking the distal end of the umbilical vein.

It would be absurd to draw any conclusions from our bacteriological findings from the small number of jaundiced babies in this series, particularly as we can demonstrate a much larger series of jaundiced babies in which blood group differences and prematurity probably accounted for a large number of the cases.

However, these observations made us alert to the possibilities of umbilical infection and have since enabled us to detect definite cases of umbilical sepsis that might otherwise have been overlooked. It seems that in time "physiological jaundice" may well disappear as a clinical diagnosis.

The danger of the hand towel as a means of spreading infection is obvious.

Some alarm has been caused by the steadily increasing incidence of resistant strains of organisms associated with the increasing popularity of antibiotics (Rountree and Thomson, 1949). Thus Barber (Barber and Rozwadowska-Dowzenko, 1948) reported that the incidence of penicillin resistance among strains of *Staphylococcus pyogenes* isolated from cases of infection had increased from 14% during a period in 1946 to 31% in 1947, and subsequently to 59% in 1948.

The concentration of streptomycin that we used in estimating the sensitivity of the organisms was a much lower one than has been used by most other investigators. This partly accounts for the high incidence of resistant strains in our series, since probably many of the resistant strains would have been sensitive to a higher concentration of streptomycin.

However, our figures confirm the high incidence of penicillin and streptomycin resistant *Staphylococcus pyogenes* in hospital practice.

This last fact, in association with the high incidence of *Staphylococcus pyogenes* generally in the nose and umbilicus of babies in the first two weeks of life, should be a strong argument against early circumcision before healing of the umbilical wound has taken place. With many patients being discharged on their seventh day from maternity hospitals, some hospitals appear to be permitting earlier circumcision, before the umbilicus has healed. This would appear to be undesirable on account of the risk of infection (Miles, Williams and Clayton-Cooper, 1944).

Summary.

1. Results of a ten-day bacteriological investigation carried out in a twenty-bed nursery in an obstetric hospital are reported.

2. Nasal and umbilical swabbings were made daily of every baby, and a high incidence of *Staphylococcus pyogenes*, increasing with the age of the baby during the first week of life, was demonstrated in each.

3. Clinical infections were noted at the same time and the clinical importance of our observations is discussed.

4. Particular stress is laid on the importance of correlating the bacteriology of the umbilicus with the clinical picture in assessing the existence of umbilical infection.

5. Penicillin and streptomycin sensitivity estimations were carried out, and a high incidence of resistant strains was demonstrated.

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IDIOPATHIC HYPERLIPÆMIA, WITH A REPORT OF TWO CASES OCCURRING IN ONE FAMILY, AND A REVIEW OF THE LITERATURE.

By PETER S. HETZEL,
Surgical Research Officer, Institute of Medical and Veterinary Science, Adelaide, South Australia.

IDIOPATHIC HYPERLIPÆMIA is the name given to a condition of unknown ætiology in which, under a variety of conditions, there is an unusually high level of neutral fat in the serum—so much so that the serum presents a milky appearance. In mild degrees of hyperlipæmia, in which the neutral fat is increased from 50% to 150%, the serum is not milky. Thannhauser (1950) stresses that the term hyperlipæmia should be reserved for conditions in which the neutral fat content of the serum is raised, as distinct from increase in cholesterol as found in hypercholesterolæmia. In idiopathic hyperlipæmia the neutral fat content alone is increased. The lecithin content may be increased, but not proportionately, and generally the sphingomyelin and cephalin fractions are not raised.

A number of cases of this condition has been reported in the last twenty years, particularly from America. In this article it is intended to consider the possible causes of hyperlipæmia, review the literature on idiopathic hyperlipæmia and report a further two cases.

Types of Lipæmia.

The fat content of the serum is influenced by a number of different factors, as put forward by Thannhauser: (i) the absorption of fat from the intestine, (ii) the migration of fat from depots to organs of fat disintegration, (iii) the deposition of fat in subcutaneous and other fat-storing tissues, (iv) the speed of fat disintegration in the organ—that is, the balance between fat transport and fat combustion, (v) disturbance of lipid metabolism within the cell.

Some of these states are found to occur independently, while others arise secondarily to some other metabolic disturbance. It is not feasible to discuss each of them in detail, but a brief consideration may be of value.

Absorption of Fat from the Intestine—Alimentary Hyperlipæmia.

Alimentary hyperlipæmia does not appear to be associated with any specific disease state. Its presence is generally shown by a fat-tolerance curve. The maximum level of neutral fat in the serum found in this test depends on the fasting level, so that diagnostically it has little value. Whether alimentary hyperlipæmia is an entity remains to be proved, for it is not considered feasible that fats can be absorbed more rapidly. Thannhauser considers it more likely that persistent alimentary hyperlipæmia is the result of sluggish deposition of fatty substances from the capillaries into the organs of fat metabolism and storage.

Migration of Fat from Fat Depots to the Organs of Metabolism—Transport Hyperlipæmia.

Transport hyperlipæmia is found in a number of diseases, including the following: (*a*) lipoid nephrosis, in which it may be associated with a lowered serum protein level; (*b*) disturbances of carbohydrate metabolism, such as von Gierke's disease and uncontrolled severe *diabetes mellitus*, secondary to decreased activity of the liver in metabolism of carbohydrate.

Transport hyperlipæmia appears to be a secondary condition.

Defective Removal of Fat from the Blood and Sluggish Deposition in Fat Depots—Retention Hyperlipæmia.

Retention hyperlipæmia comprises the third and fourth of Thannhauser's categories. After normal fat digestion fats are absorbed by two routes, some directly into the portal blood, but most reaching the systemic circulation through the lymphatics and the thoracic duct, which means that the liver is temporarily by-passed. Some of the fat is removed from the blood directly by the fat depots. The liver cells themselves have been found to contain more neutral fat than the fat depots, so that in retention hyperlipæmia there may be a disturbance both of the fat depots and of the liver.

That idiopathic hyperlipæmia falls into this category was first suggested by Holt and his co-workers (1939) when they studied their case very closely over a long period of time. They noticed that fluctuations occurred in the degree of lipæmia, and that these were often paralleled by the size of the liver. The pathological basis of the disturbance remains obscure.

Disturbances of Cellular Lipid Metabolism.

In conditions such as Gaucher's disease and Niemann-Pick's disease hyperlipæmia has not been found, and often all the serum lipid contents are normal. There has been no description of milky serum in these conditions.

Clinical Features of Idiopathic Hyperlipæmia.

The first case of idiopathic hyperlipæmia was described in 1932 by Buerger and Gruetz under the title of idiopathic hyperlipæmia with hepato-splenomegaly and secondary xanthomata. Since then 17 further cases have been reported. As described they fall into two main categories.

1. Children. In these cases a familial incidence has generally been demonstrated. The case of Buerger and Gruetz was that of an eleven-year-old boy, in whose picture hyperlipæmia was the main feature. In the case of a boy of two years secondary xanthomata were predominant.

2. Adults. In cases among adults a familial incidence has only rarely been shown; but the absence of a familial incidence among adults does not generally preclude the condition being hereditary and possibly congenital. Secondary eruptive xanthomata have been seldom reported.

In the cases reported only two patients were female, and these were children. The youngest patient was aged one year and the oldest forty-seven years.

The present patients, while adults, are members of one family. The clinical features described in the reported cases are as follows: eruptive xanthomata of the skin; variable degrees of enlargement of liver and spleen; *lipæmia retinalis*; rare attacks of abdominal pain; occasional glycosuria.

Skin.

Eruptive xanthomata have been found to develop rather suddenly on the limbs, the back and the buttocks. The size varies, but they are generally found to be small—of the order of a pin's head. They appear as discrete yellow nodules surrounded by a small reddened area. Appearance and disappearance are said to depend on the level of fat in the serum. However, in the individual case even when the serum lipid content is very high, xanthomata may not be present. The xanthomata of hypercholesterolæmia, in contrast, change very little and are present independently of any hyperlipæmia.

Abdominal Pain.

Abdominal crises of varying types have been reported a number of times. The pain is of variable onset, often very sudden, and sometimes related to food. Vomiting may be present. Clinically the pain has been accompanied by tenderness and rigidity, which in some cases has been generalized and in others was upper abdominal. In two cases fever and pronounced leucocytosis had been present. Operation was performed on each of these patients and no satisfactory local cause could be found. In his case Holt observed that the crisis of pain was preceded by a very high level of serum lipids—eight grammes *per centum*—and then as the pain developed and the attack persisted the level fell hourly. It was found that the liver and spleen increased in size and became tender. Presumably the fat was removed by these organs.

Liver and Spleen.

The liver and spleen are variably enlarged, decreasing when the level of fat in the serum has been reduced by a diet poor in fat. In one case, the patient having died from an intercurrent infection, Chapman and Kinney (1941) found that in spite of the high level of neutral fat in the serum the neutral fat content of the organs did not exceed the normal values. The pancreas was normal. In the liver and spleen a few foam cells were seen. These findings would confirm the view of Holt and others that the hyperlipæmia is of the retention type and not associated with any definite disease of the cells themselves.

In one of the cases reported by Movitt and his colleagues (1951) a biopsy of the liver was performed after the patient had been receiving a diet rich in fat for fourteen days. The parenchymal cells were found to be filled with fat. In the sternal marrow no fat-filled phagocytes were found.

The organs are firm in consistency. Their size is considerably and rapidly reduced in response to a fat-poor diet.

Lipæmia Retinalis.

The presence of *lipæmia retinalis* appears to depend, like many of the other features, on the level of serum lipid. When the serum lipid content is above 3·5 grammes *per centum*, then *lipæmia retinalis* is to be expected according to Lepard (1944), and is not found when the level is less than 2·5 grammes *per centum*. The appearance is described as a milky edge to the retinal vessels or the "vessels appear full of milk rather than blood".

Glycosuria.

Glycosuria has been found in some cases. Its occasional presence means that in the first diagnosis care must be taken to eliminate uncontrolled severe *diabetes mellitus*, which may show hyperlipæmia and xanthomata with glycosuria.

Biochemical Findings.

The total lipid contents were estimated acocrding to the method of Matas as described by Gradwohl (1948). The fatty acid contents were estimated by a modification of a method of titration as described by Harrison (1937). The total blood cholesterol content was estimated by the method of Day and Bolliger (1930).

TABLE I.

Blood Lipids in the Cases Reported in the Literature.

Author.	Total Lipid Content.[1]	Total Fatty Acid Content.[1]	Neutral Fat Content.[1]	Total Cholesterol Content.[1]	Free Cholesterol Content.[1]	Total Phosphatid Content.[1]
Buerger (1932)	9476	—	—	686	315	1740
Opitz (1935)	1800	900	—	263	165	358
Holt (1939)	7370	—	—	330	127	430
Franklin (1937)	3540	—	—	188	—	—
Bernstein (1939)	7430	—	5430	1059	677	941
Goodman (1940)	3954	3115	2775	379	158	465
Levy (1946)	—	—	—	250–329	—	—
Shore (1947)	3480	—	—	345	—	280
Bloomfield (1947)	—	—	—	864	—	—
Harslöf (1948)	3840	2671	—	596	204	573
Hopgood (1948)	—	3370	3202	250	150	328
Thannhauser (1950)—						
1	—	5196	4477	693	323	810
2	—	2994	2342	525	221	563
3	—	3370	3202	250	150	328
4	—	7200	6570	900	403	400
Movitt (1951)—						
1	3800	—	—	421	77	—
2	1375	—	—	374	—	629
3	1060	—	—	302	165	365

[1] Milligrammes *per centum*.

The results of various estimations in the reported cases are shown in Table I. It can be seen that the significant changes are in the total lipids, owing to an increased neutral fat content. In some cases the total cholesterol level is raised significantly and sometimes the total phosphatide content is increased, but the consistent finding is an increase in neutral fat content. (As the methods of estimation vary from one report to another it is not possible to give a common normal range.)

Report of a Case.

L.A.B., a married man, aged twenty-three years, was found to have lipæmic serum when he offered himself as a blood donor. A hæmaglobin estimation by a colorimetric method had given the value 140% (Sahli). He gave no history of previous illness either as a child or in later life. He had served for two years in the navy, and was discharged fit. No history of skin lesions or recurrent attacks of abdominal pain could be discovered. His father died of "heart failure" at the age of forty years, and his mother of pneumonia and pleurisy at the age of twenty-eight years. His sister and four brothers were alive and well.

On examination, the patient was a thin, slightly built man who appeared in perfect health. His skin was clear. The significant clinical findings were as follows. His weight was nine stone seven pounds. Examination of his heart showed that the apex beat was palpable three and a half inches from the mid-sternal line. There was no disturbance of rate or rhythm. No murmurs were audible. His systolic blood pressure was 120 millimetres of mercury and his diastolic blood pressure 90 millimetres. The *fundi oculorum* appeared pale; the vessels were slightly pale, but not milky. The liver was just palpable under the right costal margin and the spleen was enlarged to two fingers' breadth below the left costal margin. The consistency of both these organs was firm. They were not tender. Examination of the urine revealed no albumin, sugar, bile pigment or salts.

X-ray examination of the pelvis, chest, long bones and skull revealed no abnormality.

A blood examination gave the following findings. The red cells numbered 4,550,000 per cubic millimetre. There was a slight increase in the red cell diameters. The volume of packed cells was 45%. The mean corpuscular volume was 84. Platelets were plentiful. Leucocytes numbered 8100 per cubic millimetre, of which 64% were polymorphonuclear cells, 22% lymphocytes, 6% monocytes and 8% eosinophile cells. Slight anisocytosis was present in the red cells. A slight "shift to the right" was detected in the polymorphonuclear cells. The serum was milky.

A number of biochemical investigations were made. The total serum lipid content was 4540 milligrammes *per centum* (normal, 450 to 1260 milligrammes *per centum*); the free fatty acid content was 260 milligrammes *per centum* (normal, 190 to 640 milligrammes *per centum*); the total cholesterol content was 130 milligrammes *per centum* (normal, 100 to 200 milligrammes *per centum*); the neutral fat content (and phospholipid content) by subtraction was 4150 milligrammes *per centum* (normal neutral fat content, 0 to 200 milligrammes *per centum*; phospholipid content, 150 to 250 milligrammes *per centum*). The icteric index was four units. The serum alkaline phosphatase content was 10·0 King-Armstrong units (normal, 3 to 13 units); the serum colloidal gold, 3 (normal 0 to 2); the cephalin cholesterol flocculation test produced a very weakly positive result in forty-eight hours. The total serum protein content was 5·9 grammes *per centum* (normal, six to eight grammes *per centum*). The fasting blood sugar content was 0·09%. The glucose and galactose tolerance curves were normal. Examination of the serum under dark-ground illumination revealed a field crowded with chylomicrons exhibiting Brownian movement.

TABLE II.

Biochemical Estimations on the Serum of the Patient and His Four Brothers.

Subject.	Serum Levels in Milligrammes *per Centum*.			
	Total Lipids.	Free Fatty Acids.	Neutral Fat (*plus* Phospholipids).	Total Cholesterol.
A.	200	—	—	57
G.	190	—	—	68
H.	4900	—	—	217
L.	4540	260	4150	130
R.	300	—	—	87

All biochemical investigations were started with the patient in the fasting state.

During the period of observation, five months, the patient has remained well. He has had no symptoms of any kind. He is not accustomed to taking a diet rich in fat and he was not advised to change his dietary habits in any way. However, his liver and spleen are no longer palpable. His fundi have not changed. An estimation repeated three months after the first one gave the total serum lipid content as 3200 milligrammes *per centum*.

The patient has four brothers and one sister living. As his family lives a considerable distance from the patient it was not possible to examine them clinically, but through the courtesy of their local medical practitioner fasting specimens of serum from the four brothers were obtained. In Table II the results of the respective estimations are compared with those of the patient. Thus it appears that the patient's brother H. has a similar condition establishing a familial incidence. He is reported to be in good health.

Discussion.

Whilst the number of cases reported has been very few and only one subject has been observed *post mortem*, it seems reasonable to assume that idiopathic hyperlipæmia is a benign condition in which the prognosis is uniformly

good. The main disabilities from the patient's personal standpoint are the possible attacks of abdominal pain and the eruptive xanthomata. It seems that in some cases in which these symptoms and signs develop they recur at intervals, but the person who is untroubled by them when first examined appears to remain free.

Treatment of the condition has received a considerable amount of attention. Holt and his colleagues carried out a number of procedures in an attempt to reduce the lipæmia. Thyreoid therapy, insulin therapy and blood transfusion were tried without success. The only method of treatment which has met with uniform success is the taking of a diet poor in fat.

In a condition which has no known pathological consequences and appears as mild as this one does, it is questionable how much treatment is required so long as the patient remains free from the distressing abdominal crises and the skin lesions. The outlook is entirely different from hypercholesterolæmia, with its attendant evils. Hyperlipæmia as such does not appear to give rise to any life-shortening process within the body. On this account, apart from being advised of the nature of his condition, the patient has been told to carry out his normal activities and to eat his normal diet. He has been warned and advised of the possibilities of attacks of abdominal pain and the development of xanthomata.

Summary.

The possible causes of hyperlipæmia are considered in relation to a possible basis for idiopathic hyperlipæmia. The literature dealing with the clinical and biochemical findings in the reported cases is briefly reviewed. The symptoms of abdominal pain and the signs of eruptive xanthomata, enlarged liver and spleen, *lipæmia retinalis* and occasional glycosuria are described in detail. The reported findings in patients of an increase in total serum lipid content with milky serum, due to increased neutral fat content, are tabulated.

One case is reported in detail, and the occurrence of the condition in another of the patient's four brothers is recorded. The condition in these cases appears to be symptomless, and from investigations carried out in one of the cases, benign.

The treatment by diet and its purpose in regard to prognosis is discussed.

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SPLITTING THE AORTA AND DISSECTING ANEURYSM.

By J. B. CLELAND, C.B.E., M.D.,
Emeritus Professor of Pathology, University of Adelaide.

IF the wall of the thoracic aorta is grasped by catch forceps from its inner aspect, after one or two attempts it will be found that the media readily strips along a plane (or planes) which is nearer the periaortic coat than the intima. Sometimes this outer portion of the media is so thin that it looks as if the periaortic tissues only had been separated; but microscopic examination of sections shows the outer part of the media, which seems to be firmly anchored to the periaortic tissues. Once started, the stripping takes place with ease, particularly in lateral segments, but may be extended longitudinally throughout the aorta to the bifurcation. This tendency to split can be demonstrated in the stillborn infant and at all ages thereafter. One of the best examples was in a girl, aged ten years, who had died from encephalitis. If the plane of stripping is examined during its progress, occasional thread-like strands will be seen before they break, passing between the two layers. I think there can be no doubt that in dissecting aneurysm the blood traverses this plane (or one of these planes). The "lacings" one can see in some good examples of dissecting aneurysm passing as threads between the walls are presumably the same structures as the strands in the artificially produced condition, and may represent, originally at any rate, *vasa vasorum.*

Examination of sections from both young and old shows no change in appearance of the media ahead of the plane that is being stripped. I have never been able to satisfy myself of the existence of medial necrosis in the subjects of dissecting aneurysm that I have examined. Since the splitting readily occurs in children as well as in adults, a pathological change can hardly be held responsible. Once blood has reached the plane of splitting, and perhaps there is more than one, the dissection of the media can take place with ease. However, an explanation is still required for the tear or rupture of the intima and part of the media till this plane is reached. As neither atheroma nor syphilitic aortitis necessarily plays a part in the genesis of dissecting aneurysm, and as the rupture is usually in the ascending aorta, it seems to me more likely that a mechanical rather than a pathological factor is responsible. I have recorded the case of a young airman who was killed by being run over by a taxi-cab (Cleland,

1944); no ribs were broken, but both the aorta and the pulmonary artery were severed transversely as if by a knife. I presume that he had been caught underneath the vehicle and his chest compressed at the same time as the two large blood vessels had been filled by the systolic contraction of the heart. Perhaps some analogous sudden increase of pressure on the aorta just as it fills may start a tear—much as a sudden tap on a watermelon may split it. The tendency for the inner part of the media with the intima to tear laterally in segments when grasped by the catch forceps and pulled may also be a factor in facilitating the original tear.

In seeking for a possible useful purpose to be served by the media's consisting of two or perhaps several layers, it may be suggested that this is of advantage as the aorta becomes tensely filled with blood. By the inner layer's sliding to some extent on the outer layer, the pressure is probably somewhat dissipated and, moreover, the periaortic tissues will be less disturbed.

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Reports of Cases.

A CASE OF DEATH FOLLOWING SECTION, LIGATION AND INJECTION OF VARICOSE VEINS.

By A. E. COATES,
Melbourne,
WITH A REPORT ON THE POST-MORTEM EXAMINATION
BY K. M. BOWDEN,
Melbourne.

Clinical Record.

(A.E.C.)

MRS. H.F.H., aged forty-three years, was operated upon by me for a cerebral tumour in 1935. She had previously undergone a subtemporal decompression by Sir Alan Newton. Amputation of the left frontal lobe was performed in two stages. The tumour was partly calcified and regarded as an intracerebral meningioma. She was then a maid at the Royal Melbourne Hospital and had suffered much from headache, papillœdema and vomiting. Her memory was complete. She married a farmer, had three children and was normal in every way. In 1947 she was involved in a motor-car accident, striking her head on the windscreen. Headaches worried her, and it was thought by her doctor that some intracranial recurrence might have been the cause. However, all investigations gave negative results. She had some gynæcological disorder which was duly treated.

In March, 1951, she consulted me about varicose veins in both legs. She stated that after one of her confinements ten years previously her left leg had been swollen. Examination revealed severe varicosities in both legs, in the long saphenous veins, and there was a pigmented area about the left ankle. Tests indicated incompetent long saphenous veins. Ligature and section of these veins was advised.

On March 30, under local anæsthesia, section of both long saphenous veins was carried out, (*a*) just below the junction with the femoral vein, (*b*) above the knee, (*c*) below the knee. "Ethamolin" was injected into each long saphenous vein in the lower third of each leg—two millilitres into each side. The patient was allowed to walk at once and attended to her ablutions. Next day, while at the toilet, she suddenly died. Because of the peculiar circumstances I requested the coroner to investigate the case, and Dr. K. M. Bowden performed a post-mortem examination, of which the report appears below.

Comment.

The leg in which the patient had presumably had a venous obstruction ten years before provided the pathological setting for this tragic complication. I have long since given up the injection of sclerosing substances into the upper part of the veins after ligature and section, but now will never again use a sclerosing solution even below the third site of ligation and section—that is, in the leg—in such a case when there is a previous history of deep venous obstruction.

These "slings and arrows of outrageous fortune" serve to keep one humble. Even the simplest routine surgical procedure is fraught with some risk.

Post-mortem Examination.

(K.M.B.)

On April 2, 1951, at the City Morgue, Melbourne, I made a post-mortem examination on the body of a woman, whose age was said to be forty-three years.

The body was that of a middle-aged woman, well built and well nourished. The hair was cut short and clipped. There was an old surgical scar in the left fronto-temporal area above and in front of the left ear; it was horseshoe-shaped, as for a surgical osteoplastic flap. Some skull bone, chiefly in the left frontal region, had been surgically removed over an area two inches in diameter. Surrounding this, and one inch away from it, an osteoplastic flap had been previously cut; for this there was a series of trephine holes that had been joined together by a Gigli saw cut. The flap of bone had been replaced and had firmly united again between the trephine holes. The areas in the skull where bone was missing were filled in with a dense layer of fibrous tissue, made up of *dura mater* and membranes on the deep side. Two strands of frontal lobe tissue were adherent to the under surface of the membranes in the main deficiency in the frontal bone.

There were a number of recent surgical incisions along the course of each saphenous vein on the legs. Each saphenous vein had been cut and tied close to its insertion into the femoral vein. On the left side there were some hæmatomata in the subcutaneous tissues and some ante-mortem clot in the saphenous vein for two inches distal to the ligature, and in the small tributaries in Scarpa's triangle extending out for a maximum distance of two inches from the cut. There were three surgical incisions horizontally situated on the left leg, one near the medial condyle of the femur, another one inch below the knee joint, and a further one three inches below the knee joint. The great saphenous vein for its full length contained recent clot, which was dark red and non-adherent. It was ante-mortem clot. It was present in the saphenous vein as far as it was traced down towards the ankle. The posterior tibial veins on the left side contained similar ante-mortem blood clot. There were the remains of blood clot lightly adherent to the lining of the femoral vein for a distance of two inches immediately beyond its origin. In this area the endothelium of the vein presented the appearance of recent clot having been present, but having separated. There was no clot in the deep femoral vein. Two inches below the right knee there was another surgical horizontal cut where the saphenous vein had been ligated. The saphenous vein had also been ligated opposite the right medial femoral condyle. In the right leg a little ante-mortem clot was present in the saphenous vein adjacent to the point of ligation and distal to it for about one inch. The vein below that was collapsed and empty. There was no clot in the posterior tibial veins on the right side; there was no clot in the femoral vein, the superficial femoral veins or the deep femoral vein. The iliacs contained no ante-mortem clot.

Surrounding the left medial malleolus there was an area about two inches in diameter where the skin was dark brown and the epidermis was shed. This was an area of incipient varicose ulceration. *Pes cavus* was present on both sides with flattened transverse arches. There were many varicose veins along the distribution of the saphenous veins on the inner sides of the calves. There was an old appendiceal scar.

The pericardial sac was clear. The heart weighed 12 ounces. The left ventricle was a little dilated. The coronary vessels were patent. The muscle appeared normal. Coiled up in the pulmonary artery and its main branches there were four pieces of worm-like ante-mortem clot. Clot also extended along the pulmonary artery and its branches through the root of the left lung for a short distance into its substance.

There was an area of hæmorrhage, peripherally situated, in the upper lobe of the right lung, along the anterior margin about half-way down its length. This was in the substance of the lung for a depth of about half an inch. The lungs were rather bloodless and light.

The liver was normal. The gall-bladder was distended with dark bile; no calculi were present. The pancreas and suprarenals were normal. Examination of both kidneys showed multiple tiny adenomata scattered over the surfaces; these were adherent to the capsules and pulled out easily when the capsules were stripped off. The uterus was a little enlarged. The endometrium was soft, congested and swollen, and free blood was present in the uterine cavity (menstruation). There was a small right ovarian simple cyst, about three-quarters of an inch in diameter. The stomach contained a little fluid, no food; there was no characteristic odour. The intestine was normal.

The right side of the brain was normal. A large part of the left frontal lobe was missing anteriorly. It had been surgically removed, a cavity being left which extended well into the frontal lobe as though a tumour had been previously enucleated completely from its substance, a partial covering of frontal tissue being left about it. Examination of the brain substance around the margins of the cavity showed yellowish-brownish areas of discoloration and softening. There was no evidence of any cerebral tumour. The remainder of the brain appeared normal.

Death was due to pulmonary embolism following the surgical procedure on the veins.

A CASE OF TUBERCULOSIS OF THE SKIN TREATED WITH POTASSIUM PARAAMINOSALICYLATE (PAS).

By F. BAUER,
Repatriation General Hospital, Heidelberg, Victoria.

THE patient, aged forty-four years, who had been a prisoner of war in Malaya from 1942 to 1945, gave the following history. As far as he could remember, a rash started on the back of his right hand and over the knuckles in 1944 or early in 1945. He was treated for scabies, but this did not affect his skin lesion. He also suffered from tropical sores. After his return to Australia from Malaya in 1945, a routine X-ray examination of his chest revealed tuberculosis of his right lung, and sputum tests revealed tubercle bacilli. The rash on the back of his right hand spread slowly until June, 1950, and gradually took on the appearance of "warts". They were dry and raised about one-sixteenth of an inch. The horny tops of the lesions came off at times.

On examination of the patient on June 27, 1950, the distal and ulnar part of the dorsum of the right hand and a small adjacent part of the middle finger were involved. The lesion, which measured 6·0 centimetres by 3·5 centimetres, consisted mainly of atrophic skin and had a scalloped outline. The active edge of the lesion on the radial and adjacent proximal border consisted of a verrucose plaque measuring 5·0 centimetres in length, which was raised about 2·0 millimetres and was approximately 1·2 centimetres wide. The ulnar border was formed by a similar, smaller, active lesion measuring 2·0 centimetres by 1·3 centimetres. Finally, a small plaque 0·8 centimetre in diameter was situated over the metatarsal head of the little finger (Figure I). Apple-jelly nodules could not be demonstrated on diascopy, but greyish-yellow foci were noted in the plaques.

FIGURE I.
Photograph of the right hand taken early in the course of PAS therapy (July, 1950).

The appearance of the skin condition was considered to be consistent with the diagnosis of *tuberculosis verrucosa cutis* (Riehl and Paltauf), and to have followed inoculation through infected sputum from his tuberculous lungs.

FIGURE II.
Photograph of the right hand taken seven months after completion of the course of PAS (April, 1951).

Examination of a biopsy specimen which was taken from the involved area revealed pronounced thickening of the epidermis, due to some hyperkeratosis, and pronounced acanthosis with elongation and branching of the rete pegs.

The dermis was diffusely infiltrated with a granuloma (Figure III), in which tubercles with epithelioid cells, lymphocytes and giant cells were demonstrated (Figure IV). An abscess filled with polymorphonuclear cells was also noted in the dermis.

The Mantoux test in 1945 had given a positive result in the first strength of 0·00002 milligramme of purified protein derivative. In November, 1950, the Mantoux test was repeated, unfortunately with old tuberculin, and found to give a positive result with 1:10,000 of old tuberculin.

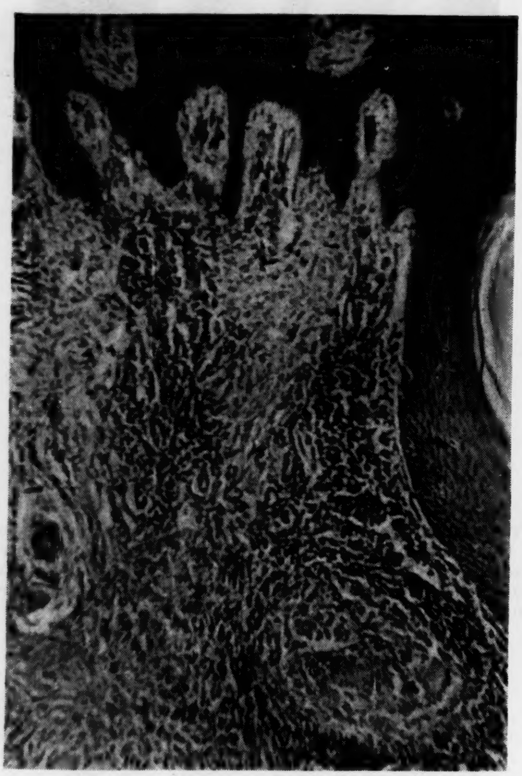

FIGURE III.
Tuberculosis verrucosa cutis (x 100). Pronounced acanthosis of the epidermis and an infiltrate in the dermis with a typical tubercle in the right hand corner may be seen. (Half the thickness of the epidermis had to be excluded from the photograph to include this tubercle.)

An X-ray film of the chest, taken in 1945, revealed a cavity at the right lung apex and some infiltration at the left apical area.

Right pneumothorax was induced on November 30, 1945, and refills were kept up until January 21, 1947. It was noted for the first time on February 22, 1947, that the cavity in the right apex had closed. The lesions in both lungs remained unchanged until June, 1949, when X-ray pictures revealed a spread of the lesion in the upper lobe of the left lung. A further deterioration took place in November, 1949. Because of this unstable lesion in the left lung, a course of PAS was commenced in June, 1950. X-ray films of the chest, which were taken after the PAS course had been completed, revealed some improvement in the condition of the left lung, and a more recent X-ray film on March 28, 1951, showed that this improvement had been maintained.

Sputum tests which were made on two separate occasions in 1945 revealed acid-fast bacilli. Further repeated examinations did not reveal tubercle bacilli.

The Wassermann test of the blood, which was carried out towards the end of 1950, gave negative results.

As the patient, when first examined, was already having a course of PAS for his chest complaint, it was decided to postpone the giving of calciferol or streptomycin for his skin lesion until later, and he was observed for further progress.

The dosage of the course given consisted of 12 grammes of PAS per day, given in four equally divided doses for six days per week, and the course lasted for twelve weeks, from June 26 to September 17, 1950.

The skin lesions were watched and their appearance improved considerably during the course of treatment. A note was made in October, 1950, that the lesions at that time involved the same area as before, and consisted mainly

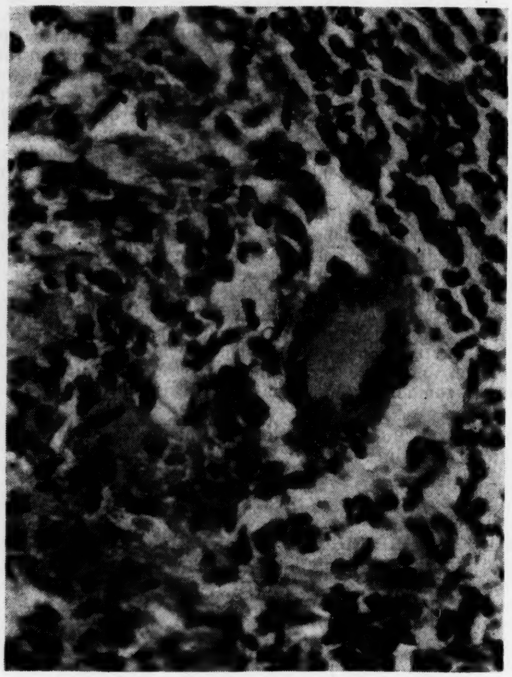

FIGURE IV.
High-power photomicrograph (x 500), showing a higher magnification of part of the tubercle in Figure III.

of purplish atrophic skin. Some infiltration may have been present in the most proximal edge of the lesion, and on the middle finger knuckle was a very small verrucose area. At the end of December the skin appeared to be free of active disease, and all that remained to be seen was slightly atrophic purplish-colourd skin in the area previously involved. The appearance remained unchanged when the patient was last examined in June, 1951 (Figure II).

Examination of a biopsy specimen, which was taken in April, 1951, revealed thin epidermis with the rete pegs flattened in part of the section and absent in others. The dermis was thin and the collagen slightly denser than normal, with a moderate degree of small round-cell infiltrate in the upper part of the dermis (Figure V).

Comment.

Tuberculosis verrucosa cutis is a rare disease in this country, and this condition is not much influenced by sanatorium type of treatment alone. This patient was under such general treatment for four years without diminution in the tuberculous lesions of either his lung or his skin. However, after the institution of PAS therapy for his pulmonary condition, whilst there was only a

moderate improvement in his lung condition, there was a dramatic remission in his skin tuberculosis.

Summary.

1. A case is described in which potassium PAS therapy, given for pulmonary tuberculosis without much effect on the pulmonary lesions, led to the apparent cure of associated *tuberculosis verrucosa cutis*. This case is the first to be reported in this journal in which the diagnosis has been supported by confirmatory pathological and microscopic investigations.

2. As PAS is used with some success in tuberculosis of the lungs, and as reports in respect to its use in skin tuberculosis are not forthcoming, this case is published on account of the good response to PAS in this case of skin tuberculosis. One cannot draw any conclusions from one successful case, in which the follow-up has not been sufficiently long, therefore no claim is made for the efficacy of the treatment. However, it is suggested that PAS may be tried when skin tuberculosis does not respond satisfactorily to standard therapy.

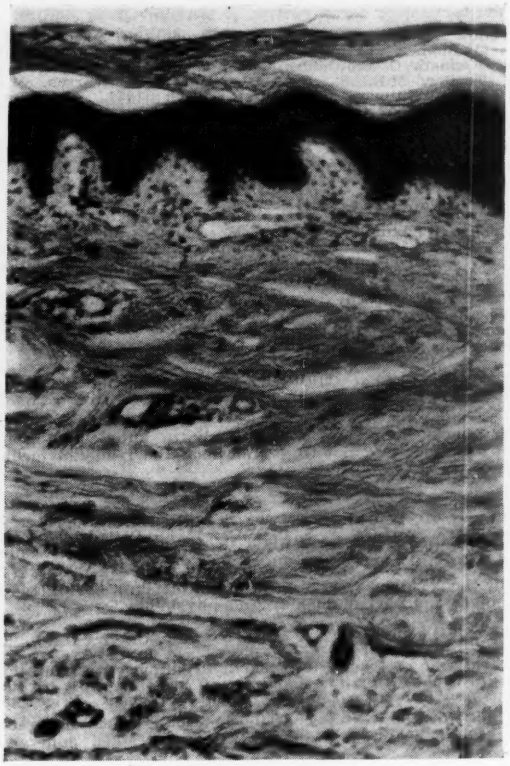

FIGURE V.

Low-power photomicrograph (×100), showing thin epidermis. The tuberculous infiltrate in the dermis has disappeared.

Acknowledgements.

I wish to express my thanks to the Chairman of the Repatriation Commission for permission to publish this article; to Dr. W. L. Forsyth, specialist medical officer, chest diseases, under whose care this patient was admitted to hospital; to Dr. K. G. Colquhoun, visiting specialist, dermatology, for his advice; to Dr. R. B. Maynard, senior specialist medical officer, pathology, for assistance from his department; and to Miss H. Wischusen, pathological technician, for taking photographs and cutting the sections.

Reviews.

IMMUNOLOGY.

A BOOK of 700 pages on immunology has been prepared for "medical students and others who have had training in pathogenic bacteriology, inorganic and organic chemistry, and who are interested in the underlying principles involved in infection, resistance and diagnostic laboratory tests. It has been in use in mimeograph form in the Universities of Kansas and Montana for the past few years".[1] The author, N. P. Sherwood, Professor of Bacteriology in the University of Kansas, has obviously planned it to meet his own course of instruction. Actually the field covered is much wider than a strict attention to the title would permit, and the author's admirable wish to develop the historical approach is a large factor in this.

The chapter headings indicate the plan to consider infectious agents, the host-parasite relationship, the mechanism of cellular defence and their anatomical relationships before immunity, natural, acquired and experimental, is discussed. Antibodies and their production, measurement and use are described in a section of some 200 pages; antigens on the other hand are condensed into less than 100 pages. There is a useful chapter recapitulating the facts concerned with specificity before the author expands largely on the complement-fixation tests for syphilis. The last 200 pages are chiefly occupied with a consideration of hypersensitiveness and the tuberculin reaction. The last chapter is an appendix of definitions of physical chemistry in the descriptive manner.

Each chapter is followed by a comprehensive list of references and a supplementary list also. There are constant exhortations in the text that the student should read the original papers, and one is compelled to reflect that American students must spend an enormous amount of time in the libraries if they heed Professor Sherwood!

Criticisms of the volume are mainly in the direction of its scope. One wonders whether the student needs to have at hand the method of preparing tuberculin fractions, or the details of the complicated and specialized testing of the Rh factors in human blood, or suggestions in regard to surgical treatment of wounds and fractures along with the indications for tetanus prophylaxis.

There are a few printer's errors; "cycle cell anæmia" lingers in the mind. Paper and type face are admirable, and for the laboratory worker, the book has the advantage of opening and lying flat at the page chosen.

This book seems too comprehensive for the British standard of undergraduate teaching, which attempts to make the student concentrate on principle, and avoids burdening him with techniques at which he will never become expert. Probably this book will find its best place as a reference book in clinical laboratories.

GROWTH AND DEVELOPMENT OF CHILDREN.

E. H. WATSON AND G. H. LOWREY, of the University of Michigan Medical School at Ann Arbor, have produced a valuable book entitled "Growth and Development of Children".[2] It is a careful selection of factual data from far and wide on many diversified aspects of the subject. Very little attempt has been made to provide any critical assessment of the matter selected. As the decision to include or to exclude material has been difficult, it is apparent that inclusion carries with it the approval of the editors. An effort has been made with success to present the facts graphically by the free use of tables, charts and outlines to save space and to achieve definition and clarity. Diagnostic and therapeutic procedures have been deliberately omitted. References have been selected with great care to allow interested people to find how the facts can be usefully applied in actual practice and to amplify the information supplied in the book itself.

A neat distinction has been drawn between "growth" and "development". "Growth" is used to mean an increase in

[1] "Immunology", by Noble Pierce Sherwood, Ph.D., M.D., F.A.C.P.; Third Edition; 1951. St. Louis: The C. V. Mosby Company. Melbourne: W. Ramsay (Surgical) Proprietary, Limited. 8½" × 5½", pp. 732, with 7 plates in colour and 21 figures. Price: 84s.

[2] "Growth and Development of Children", by Ernest H. Watson, M.D., and George H. Lowrey, M.D.; 1951. Chicago: The Year Book Publishers, Incorporated. 9" × 6", pp. 260, with 54 figures. Price: $5.75.

physical size of the whole or any of its parts; it can be measured in terms of length, weight or metabolic balance. "Development", on the other hand, is used to indicate an increase in skill and complexity of function; the individual develops neuro-muscular control, dexterity or character.

An indication of the scope of the book can be gained from the information that the growth periods covered include three pre-natal periods, prematurity, birth, neonate, infancy, pre-school, pre-pubertal and adolescent periods. No longer do pædiatricians concentrate on mere physical measurements, but they are taught to measure everything associated with growth and development as defined above; there are whole chapters on the assessment of behavioural development, organ development and energy metabolism. The twelfth and final chapter is devoted to some relevant aspects of abnormal growth, a consideration of which has assisted the students of normal growth. In a chapter of only twenty-three pages, we find a remarkably satisfactory exposition of the role of the endocrine glands in normal growth and development. Scattered through the book there is information on embryology and phylogeny and on heredity which it would be hard to get so succinctly elsewhere. Abstruse mathematical calculations have been avoided to keep the book small and readily comprehensible by mere doctors and medical students. It should find a place in every medical library as a valued reference book.

SENSORI-MOTOR FACTORS IN DISTURBANCES OF EQUILIBRIUM.

PROFESSOR L. HALPERN is professor in the Faculty of Medicine in the Hebrew University of Jerusalem and has been investigating for a number of years the sensori-motor, mostly sensory, factors in disturbances of equilibrium. In his book, which is largely occupied with case histories illustrating his opinions, he epitomizes the results of his researches.[1] The author is one of those who are so occupied with their observational or experimental activities that they pay inadequate attention to investigations carried out in other countries. The name of Sherrington is absent and little reference is made to the proprioceptive system. The regulation of equilibrium, according to this author, is controlled by the cerebellum and the frontal cerebral cortex, and hence the fronto-cerebellar tract is of high importance. Lesions of the cerebellum produce ipsilateral disturbances, whereas those of the frontal cortex produce contralateral. Clinical evidence is cited to support the contention that primary sensori-motor symptoms in disorders of equilibrium are constant whilst secondary symptoms are variable. Sensory disturbances are regarded as playing a dominant role in disequilibration, and the author claims that this factor is of outstanding importance in practical neurology. Some of the phenomena described do not fit in completely with the author's ideas, but he asserts that his approach to the problem is new and illuminating.

Notes on Books, Current Journals and New Appliances.

MODERN TREATMENT.

As its subtitle makes clear, "Modern Treatment Year Book, 1951" deals with more than treatment alone.[2] In the preface the editor, Sir Cecil Wakeley, points out that "treatment is what the practitioner requires for his patients, but no treatment should be given until a diagnosis has been made", and this approach is adopted by the contributors. The result is advice on treatment of an essentially practical character, but based on an understanding of the patient and his affection. The 39 articles included, most of them by well-known British authorities, range over the whole field of medicine and surgery.

[1] "*Le syndrome d'induction sensorimotrice dans les troubles de l'équilibre*", by L. Halpern; 1951. Paris: Masson and C^ie^. 9" × 5½", pp. 104, with illustrations. Price: 400 fr.

[2] "Modern Treatment Year Book, 1951: A Year Book of Diagnosis and Treatment for the General Practitioner", edited by Sir Cecil Wakeley, K.B.E., C.B., M.Ch., D.Sc., P.R.C.S., F.R.S.E., F.A.C.S., F.R.A.C.S. (Honorary); 1951. London: Published for The Medical Press by Baillière, Tindall and Cox. 9" × 6", pp. 382, with 61 illustrations. Price: 17s. 6d.

Books Received.

[The mention of a book in this column does not imply that no review will appear in a subsequent issue.]

"Biochemical Institute Studies IV: Individual Metabolic Patterns and Human Disease: An Exploratory Study Utilizing Predominantly Paper Chromatographic Methods", from the Biochemical Institute and the Department of Chemistry, The University of Texas and the Clayton Foundation for Research, Austin; 1951. Austin, Texas: The University of Texas Publication. 10½" × 7", pp. 206, with about 30 text figures. Price: $1.00.

The title is self-explanatory. Bonafide investigators may receive copies gratis by applying to the Biochemical Institute, The University of Texas.

"Yellow Fever", by George K. Strode, M.D., editor, and John C. Bugher, M.D., J. Austin Kerr, M.D., Hugh H. Smith, M.D., Kenneth C. Smithburn, M.D., Richard M. Taylor, M.D., Max Theiler, M.R.C.S., L.R.C.P., Andrew J. Warren, M.D., and Loring Whitman, M.D.; First Edition; 1951. New York: McGraw-Hill Book Company, Incorporated. 10" × 7½", pp. 726, with 77 illustrations. Price: $9.50.

Describes the studies, experiments and methods of control of yellow fever undertaken and developed by the Rockefeller Foundation.

"Spleen Puncture", by Sven Moeschlin, translated by A. Piney, M.D.; 1951. London: William Heinemann (Medical Books), Limited. 10" × 6½", pp. 240, with 55 illustrations. Price: 30s.

The purpose of the book "is the discussion of the technique and the results of spleen puncture based on some 300 personal observations".

"Headaches: What Causes Them: How to Get Relief", by Noah D. Fabricant, M.D.; 1951. London: Staples Press, Limited. 7½" × 5", pp. 160. Price: 7s. 6d.

The book "has been written only to supplement sound medical advice and not to confer upon the reader a medical degree".

"Bases of Human Behavior: A Biologic Approach to Psychiatry", by Leon J. Saul, M.D.; 1951. Philadelphia. J. B. Lippincott Company. Sydney: Angus and Robertson, Limited. 9½" × 6½", pp. 166, with 8 illustrations. Price: 43s.

The book "has grown out of an attempt to introduce psychiatry as part of biology and physiology".

"Light Out of France: French Contributions to Civilization", edited by John G. Stanbury and A. R. Chisholm; 1951. Sydney: Angus and Robertson Limited. 8½" × 6", pp. 222. Price: 25s.

Aims to show the contribution of France to recent scientific achievements in the most diversified spheres of thought.

"Physical Diagnosis", by Raymond W. Brust, A.B., M.D., F.A.C.P., with an introduction by Truman G. Schnabel, A.B., M.D., F.A.C.P.; 1951. New York: Appleton-Century-Crofts, Incorporated. 8½" × 6", pp. 306, with 60 illustrations. Price: $4.50.

A book in which special emphasis is placed on the use of the senses.

"Syllabus of Human Neoplasms", by R. M. Mulligan, M.D., 1951. Philadelphia: Lea and Febiger. Sydney: Angus and Robertson, Limited. 10½" × 7½", pp. 318, with 230 illustrations. Price: £4 9s.

Designed to present "the pertinent pathologic facts about the more common human neoplasms together with a correlation of their salient clinical features".

"Medicine of the Year", edited by John B. Youmans, M.D.; Third Issue: 1951. Philadelphia: J. B. Lippincott Company. Sydney: Angus and Robertson, Limited. 9½" × 6½", pp. 304. Price: 53s. 9d.

The "story of medicine" for twelve months is told—included, according to the introduction, are advances, discoveries, new concepts, conclusions reached, failures and successes.

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SATURDAY, SEPTEMBER 22, 1951.

DESCARTES AND MEDICINE.

IN 1650 René Descartes, French philosopher, mathematician and physiological theorist, died in Stockholm from a lung infection. He is best known today for his contribution to philosophy, but it is interesting to note that he wrote what Charles Singer[1] describes as the first modern book entirely devoted to the subject of physiology. Indeed, Descartes made many essays into the field of basic medical thought; yet, according to H. P. Bayon,[2] he did not succeed in making a single lasting discovery in medicine, nor did he recognize at their full value those that were being made in his time. Bayon records this assessment in a short communication on Descartes's part in the history of medicine, made last year to the Section of the History of Medicine of the Royal Society of Medicine in honour of the tercentenary of Descartes's death. In tracing the significance of this fact—if fact it is—Bayon draws attention to incidents which in 1628 led to Descartes's becoming persuaded that the problems of biology and medicine were susceptible of mathematical proof. Descartes had had no training in medicine, but from then on, Bayon states, there is frequent proof of his keen interest in medical matters. He aimed at solving all medicine's problems. A concise picture of his methods is given us by Ivor B. Hart:[3]

> He was not a member of the experimental school of Galileo and Gilbert. He had very little to do with the laboratory, except perhaps in his work on anatomy. His methods were essentially those of the mathematician. He would start with a statement of fact—an enunciation of a definite principle, and working from it, he would develop, step by step, a logical sequence of deductions until he had built up a complete scheme. He was in a sense a pioneer of the mathematical study of nature. "Comparing the mysteries of nature with the laws of mathematics, he dared to hope that the secrets of both could be unlocked with the same key."

In 1630, Bayon tells us, Descartes wrote to his old friend Marin Mersenne (who was suffering from erysipelas), telling him that he should preserve his health till he (Descartes) had discovered a system of medicine capable of irrefutable demonstration. He also wrote to Mersenne that after eleven years of study there was no portion of the body he could not explain, but as yet he could not cure even a fever. Thus his work was unavailing. It should not be thought, however, that Bayon condemns Descartes on this score. That is not his point. He comments that it is unfortunate that Descartes is more often remembered for the mistaken opinions he expressed (for example, that the pineal gland contained the soul of man) than for the useful principles he propounded, and he goes on to state that "to solve the difficulties of medicine by accurate methods was not a preposterous suggestion—it was only premature, for the necessary factual information was lacking". Borelli, who was junior to Descartes by twelve years and survived him by twenty-nine years, made "the only possible adaptation at the time"—the mechanical explanation of certain muscular movements; the same principles still find application today, and so, as Singer has stated, Borelli's achievement was more lasting than that of Descartes. Bayon sums up his view of Descartes thus:

> Therefore, though it is a misuse of words to say that Descartes contributed personally to physiological or even to pathological knowledge, yet it can be reasonably suggested that, by his advocacy of experiment as a confirmation of theory, by his attempt to make biological laws and medical observations amenable to mathematical proof . . ., by his inspiration of iatrophysics—possibly a sterile, but not a retrograde system of medicine and, by no means least, by his efforts to combine physiology with psychology in relation to reflexes—Descartes deserves to be honourably mentioned in the history of medicine.

Bayon's assessment offers a useful middle road among the widely varying views about Descartes. It is rather remarkable that students of medical history should have drawn such different conclusions about this man, who cannot be ignored in the history of thought, no matter what one's opinions of his ideas. Bayon quotes a number of well-known medical writers, whose statements we may briefly repeat. Singer has stated that Descartes's view of the nervous system and its power of coordinating the different bodily activities is "not far from the modern viewpoint, though in fact he was grotesquely wrong in detail". Osler has written of Descartes as "the first foreigner of distinction . . . to accept Harvey's views" (though it is clear that in a number of details he held erroneous views conflicting with those of Harvey). Needham has asserted that in certain of his ideas on embryology Descartes was many years before his time. Guthrie has described the effect of Cartesian philosophy on medical science as "considerable". Castiglioni has stated that Descartes's name "has great importance for the history of medicine, not only on account of his personal contributions as physiologist and pathologist, but also on account of the effect that Cartesian philosophy exerted on the evolution of medicine". Creutz and Steudel fail to mention Descartes in their history of medicine. Jefferson describes Descartes as "deeply intrigued by medicine", a statement that sounds damning for one who set out to solve the whole problem of medicine. To these opinions we may add certain others. Hart, whom we have previously quoted, calls Descartes "a very great man" and refers to "his eminent contributions to anatomy, biology, and moral philosophy". Victor Robinson,[1] in a charac-

[1] "A Short History of Medicine", 1928.

[2] *Proceedings of the Royal Society of Medicine*, November, 1950.

[3] "Makers of Science", 1923 (reprinted 1945).

[1] "Pathfinders in Medicine", Second Edition, 1929.

teristic study, states that Descartes "was too great a philosopher to be an equally great physiologist", and concludes: "He was one of the men who doubted, and thus saved the world." Perhaps a more illuminating comment on the thought that lies behind the first of Robinson's quoted statements is Guthrie's remark[1] that Descartes was "primarily a philosopher and only incidentally a physiologist"; and we shall miss the point of this remark if we do not bear in mind the fact that the philosopher in Descartes's day felt himself free to allow his thought to range in many fields that the philosopher of today would, without special training, leave alone. Descartes must, it seems, be thought of always as a philosopher and then always against the background of his own contemporary thought. In an important recent assessment of Descartes and his place in the development of modern scientific ideas, the Cambridge historian, Herbert Butterfield,[2] asserts that experiment had only a subordinate place in the system of Descartes—"he worried less about establishing a fact than about its explanation"; and, again, "the physics of Descartes . . . depends in a particular way upon his metaphysics". His importance in history, in science, in medicine, lies in his influence on the development of thought rather than in any conclusions he reached. Curiously enough, according to Butterfield, even as a thinker Descartes has been much misunderstood, and he himself complained that he was misunderstood in his own day; moreover, this very misunderstanding was important. He certainly "decided that he must sweep away all ancient opinions and start all his thinking over again"; he determined "to doubt everything and start naked once again, without any foothold whatever save the consciousness that I who do the doubting must exist—even though I may doubt whether I am doubting". But the corollary was not entirely according to his plan. For, Butterfield continues, those who never understood the positive teaching of Descartes, and who could never have risen to his philosophy, appreciated his dramatic rejection of inherited systems and ideas.

> And though he himself said that the attempt to overthrow all tradition in this way was not a thing to be carried out by any and every man; though he cautioned against any imitation of the sceptics—for, in fact, he was only doubting in order to find a firmer basis for belief or certainty—still the influence of the policy of methodical doubt was in the long run to be most significant on the destructive side and in the realm of general ideas.

His doubting was for himself and for his own immediate purposes, a matter of expediency; and willynilly he becomes the archetype of the militant sceptic. In line with his thesis that "all things which we clearly and distinctly conceive are true", he accepted that "God was another of those clear ideas that are clearer and more precise in the mind than anything seen by the actual eye" and that "everything hung on this existence of a perfect and righteous God"; and yet the agnostic justifies his agnosticism by the Cartesian system because he cannot see God. It is little wonder that varying views are held of the place of Descartes in medical thought when his essential philosophy is so differently represented. However, distorted though his views may at times be, he made a contribution to thought that carried it far forward, and for that alone science, including medical science, must be grateful. Scepticism for its own sake has little virtue and can lead to serious obscurantism; doubting "in order to find a firmer basis for belief or certainty" opens the door to the light.

[1] "A History of Medicine", 1945, page 197.

[2] "The Origins of Modern Science", 1949 (reprinted 1950), page 100.

Current Comment.

THE EFFECT ON BLOOD COAGULATION OF DRUGS USED IN CARDIAC FAILURE.

THE occurrence of thrombotic episodes in the course of cardiac failure is a matter of common knowledge, and it has been generally accepted that the mechanisms concerned are slowing of the circulation and damage of the vascular endothelium by anoxia. The great interest taken recently in the physical and chemical factors concerned in coagulation of the blood has turned attention to the possibility that some specific effect may be exerted by the drugs usually employed in the relief of congestive cardiac failure. In other words, in the treatment of heart failure is there a risk of producing an increased tendency to clotting of the blood by the agents used? Various observers have noted acceleration of thrombosis in animals after the administration of digitalis and strophanthin, and mercurial diuretics have also been studied in this connexion. Some work has also been carried out on human subjects. The latest work is by Soini A. N. Pere,[1] who set out to answer these questions: What effect have digitalis, strophanthin and "Novurit" (one of the mercurial diuretics) on blood coagulation, and what mechanisms are involved?

It should be pointed out here that, since these drugs are used chiefly on patients who have a disturbed balance and distribution of water in the tissues as a result of cardiac failure, the factor of œdema must also be taken into consideration. The author used the classical hypotheses of blood coagulation as a basis of his work, and took into consideration various factors modifying the rate of coagulation, including excitation of the vegetative nervous system. The observations were made on patients in hospital with congestive cardiac failure. Out of 105 patients, 37 were treated with digitalis alone, 27 with strophanthin alone, and 41 received injections of "Novurit" in addition to these drugs. Controls were used in practically identical numbers; these subjects were patients in the neurological wards, otherwise normal. Patients suffering from any condition which *per se* might affect the coagulation of the blood were excluded from the series. Care was also taken to ensure that no other drug was used which might have a disturbing effect on this mechanism. The types of heart failure encountered were fairly distributed among the common varieties. On admission of the patients to hospital a full clinical examination was made, and the blood was investigated to determine the histological characters, the coagulation time, the calcium time, the prothrombin index and the fibrinogen of the plasma. Standard methods were employed, and possible fallacies in prothrombin measurement were recognized, owing to the lack of full activation of prothrombin into thrombin. Technical details of the methods used in the laboratory and of the statistical analysis are omitted in this brief review. The results of Pere's observations showed that digitalis appeared to shorten the coagulation time of patients with congestive failure but not of the controls. The degree of shortening was not great, averaging $9{\cdot}8\% \pm 2{\cdot}2\%$. Strophanthin caused rather more shortening, $11{\cdot}3\% \pm 2{\cdot}7\%$; no effect was observed in the controls. It should be noted that the intravenous route was employed for this drug. The effect of the mercurial diuretic was much more striking; it was of course observed much more quickly, for after three to five hours the clotting time was reduced by $50{\cdot}4\% \pm 1{\cdot}9\%$ on an average. The figures

[1] *Acta medica Scandinavica*, Supplementum CCLI, to Volume CXXXIX.

given for digitalis and strophanthin were obtained after four to six days. These effects were much more striking in those patients from whom at the same time œdema had rapidly disappeared. "Novurit" produced some acceleration of coagulation in some controls, but in only those who had diuresis. No statistically significant change was observed in the prothrombin content of the blood of patients treated with drugs for cardiac failure or of controls. Some changes were found in the fibrinogen determinations of the patients, but these did not differ to any significant extent from the figures for the controls. Further, none of the drugs investigated had any effect on blood coagulation when tested *in vitro*. Pere discusses his technical methods, and also examines in detail the relation between the coagulation time and the secretion of urine by the patients. He concludes that it is probable that the cause of the shortening of the clotting time is to be found in an increase of free thromboplastin, which in turn is due to the passage of tissue fluid into the blood as the œdema disappears and hæmoconcentration increases. He points out that it has been shown that tissue thromboplastin is much more active than that derived from the platelets. Serial studies demonstrated the rise and fall of the coagulation time with the fluctuations of the urinary excretion, though there was also a steady and definite increase of clotting time as the congestive state decreased. Thus there appears to be no specific action exerted by the drugs tested and their effect is due rather to their diuretic action. It is helpful to have this carefully planned and evaluated work as a basis for our ideas concerning the possible causes of vascular thrombotic complications of cardiac failure, especially as the pharmacological action of digitalis seems still to be questioned.

THE ÆTIOLOGY OF PERNICIOUS ANÆMIA.

THAT pernicious anæmia is in some way a nutritional deficiency disease became evident with the demonstration by Minot and Murphy in 1926 of the effectiveness of liver feeding. The hæmatological characteristics of the disease, the frequency of glossitis and neural complications, and the almost invariable lack of gastric secretion were well known prior to this. W. B. Castle, who has done so much in elucidating the cause of pernicious anæmia and the effective treatment of the disease, has reviewed advances in knowledge of the disease in a lecture to the American College of Physicians.[1] With the advent of liver therapy it appeared that the gastric dysfunction was unique in persisting unaltered despite improvement in the patient's condition in all other respects. It was suggested that the lack of gastric juice "conditioned" a nutritional deficiency which did not exist for the person with normal gastric function. The finding that beef muscle administered with normal human gastric juice promoted hæmatopoiesis in pernicious anæmia while the beef muscle alone was without effect led to the conception of two factors, one in the food (extrinsic) and the other gastric (intrinsic). The extrinsic factor was found in many foods associated with proteins but was not itself a protein. It appeared that it might be a member of the vitamin *B* complex. None of the known members of the complex, either alone or in combination, was found to acquire increased hæmatopoietic power when simultaneously administered with normal gastric juice. While folic acid (pteroylglutamic acid) has striking effect in increasing blood formation in pernicious anæmia, it is not the extrinsic factor, for its effect is not increased by gastric juice.

The discovery of vitamin B_{12} opened the way to a clearer understanding of the ætiology of pernicious anæmia, for vitamin B_{12} and its congeners acquire increased blood-forming power when administered orally simultaneously with gastric juice. It appears, then, that vitamin B_{12} or a closely related compound in the food constitutes the "extrinsic" factor. Available facts suggest that the function of normal gastric juice is not to produce some new substance from vitamin B_{12} or the food factor, but to facilitate absorption of vitamin B_{12} from the intestine. Parenteral administration of vitamin B_{12} is extraordinarily effective in the treatment of pernicious anæmia; as little as one microgramme per day is adequate in most cases of the disease. On the other hand, the daily administration of 50 microgrammes by mouth is relatively ineffective with most patients, presumably because of the lack of "intrinsic" factor in most patients with pernicious anæmia.

The nature of the "intrinsic" factor is practically unknown. It is a thermolabile, alkali-resistant, non-dialysable substance produced in the stomach walls or parts of the wall. It has been assumed that it is an enzyme though not one of the known enzymes of gastric secretion. Evidence has been given suggesting that it is a proteolytic enzyme or a peptidase, but the evidence is not convincing, particularly when one considers the marked effect normal gastric juice has on the absorption of pure vitamin B_{12} in patients with pernicious anæmia. Many bacteria require vitamin B_{12} for maximum growth, and it has been found that normal human gastric juice and fractions of hog stomach mucosa render vitamin B_{12} unavailable for microbial growth. This suggests that the intrinsic factor enhances the effectiveness of vitamin B_{12} on oral administration by protecting it from destruction or absorption by the intestinal flora of the patient with pernicious anæmia. Other factors are also important in blood formation, and one of these is folic acid (pteroylglutamic acid). This substance in daily oral doses of one or more milligrammes produces prompt increases in blood formation and at least initial clinical improvement in all varieties of nutritional macrocytic anæmia, including pernicious anæmia, sprue, tropical macrocytic anæmia and the pernicious anæmia of pregnancy and tapeworm infestation. Some of these forms of anæmia fail to respond to vitamin B_{12}. These conditions appear to be due to a deficiency of pteroylglutamic acid or its closely related and metabolically active form, the so-called citrovorum factor. The treatment of patients with pernicious anæmia by means of pteroylglutamic acid alone often failed to produce a complete hæmatological remission or to maintain it when once achieved. Glossitis sometimes reappeared and a considerable incidence or progression of subacute combined degeneration of the spinal cord developed. The substitution of vitamin B_{12} for pteroylglutamic acid produces a prompt and complete hæmatological and clinical remission which is maintained so long as such therapy is continued. Both vitamin B_{12} and pteroylglutamic acid are required for normal hæmatopoiesis and perhaps also for the functional integrity of the gastro-intestinal tract and the nervous system in man. In bacteria evidence has been produced that these substances are necessary for the series of reactions leading to the formation of nucleic acid and the nucleo-proteins. Large daily doses of uracil or thymine (methyluracil) have been shown to be effective in certain cases of pernicious anæmia. These substances are constituents of nucleic acids.

Vitamin B_{12} is produced by a number of microorganisms and is now a by-product of the microbial production of antibiotics. It is of interest that the fæces of the patient with pernicious anæmia contain a supply of vitamin B_{12} ample to prevent the disorder if it were parenterally available to the patient. The structure of vitamin B_{12} is not yet fully known, but it is an organic cobalt compound. This is of interest in Australia because of the discovery by Underwood in Western Australia and by Marston in South Australia of a cobalt deficiency anæmia in sheep and other animals. Ascorbic acid is another substance necessary for normal blood formation. Dietary deficiency of ascorbic acid probably restricts the formation in the body of the metabolically active and closely related form of pteroylglutamic acid known as the citrovorum factor or folinic acid. When pteroylglutamic acid is fed to normal subjects, citrovorum factor is excreted in the urine. In patients with scurvy this excretion is small until ascorbic acid is given. Aminopterin when administered to pernicious anæmia patients prevents the hæmatopoietic effect of folic acid, but not of the citrovorum factor.

The full significance of the interrelations of these substances with respect to hæmatopoiesis and nucleic acid formation still remains to be disclosed.

[1] *Annals of Internal Medicine*, May, 1951.

Abstracts from Medical Literature.

MEDICINE.

Untoward Sequelæ of Sympathetic Nerve Blocks.

L. R. ORKIN *et alii* (*The Journal of Thoracic Surgery*, December, 1950), writing from an experience of 358 nerve blocks of the stellate and upper thoracic sympathetic ganglia, state that successful denervation is easily accomplished, but at a risk of undesirable sequelæ. These occurred in 21 of their patients. Traumatic pneumothorax, accidental spinal anæsthesia (the symptoms of which were alarming) and alcohol neuritis were the commonest untoward events. They state that the paratracheal approach to the cervical sympathetic is safer than lateral or posterior approaches.

Psychogenic Changes in the Field of Vision.

LOUIS J. GOGELA AND C. WILBUR RUCKER (*American Journal of Ophthalmology*, February, 1951) state that concentric contraction and spiralling of the visual fields usually associated with psychogenic factors can occur with frontal lobe tumours. The psychogenetically determined defect may be superimposed on a neoplastic or vascular disease process. Of more than 1000 patients who had tumours of the frontal lobe, 500 underwent field studies. Of this group 18 had visual fields of the so-called functional type. In 15 cases the fields were contracted concentrically, and in two cases a fatigue spiral appeared. In the remaining case there was a ring type scotoma. The possibility of the presence of an organic lesion should always be considered when psychogenic field defects are encountered.

Cortisone and ACTH.

R. H. FREYBERG *et alii* (*Annals of the Rheumatic Diseases*, March, 1951) discuss practical considerations in the use of cortisone and ACTH for rheumatoid arthritis. They state that in this condition a short course of treatment does not give good results, and there are dangers of serious side effects. In 53 cases, therefore, the authors tried the effects of intermittent treatment with cortisone and ACTH, supplementary treatment and prolonged treatment. They conclude that cortisone and ACTH do not cure any illness, though they have profound pharmacological effects. Repeated short treatments were not effective. Pregnetriolene, adenosine triphosphate, testosterone, pregnenolone and gold therapy were all undependable when used after withdrawal of the hormones cortisone and ACTH. The best treatment was to continue the use of the hormones in smaller amounts or less frequently—for example, cortisone 100 milligrammes injected three or four times weekly for up to 200 to 300 days. Relapses were common when treatment was discontinued. Cortisone was used in 31 cases and ACTH in 22. One hundred milligrammes of cortisone were injected daily on every second day at first. Gradual withdrawal was generally followed by a relapse of the arthritis. Both with ACTH and with cortisone the adrenal glands appeared to be affected adversely, since ACTH produced a poor eosinophile cell response. Tablets of 100 milligrammes of cortisone were given daily by mouth for twenty days or less to four patients with rheumatoid arthritis. Good results were obtained, which disappeared when oral treatment was suspended. Injections of 50 milligrammes of cortisone into affected joints produced a diminution of swelling, pain, inflammation and stiffness, but the disability recurred. A swollen face ("moon face") was noted in several patients treated with ACTH.

Penicillin Levels in the Blood.

A. B. CANNON *et alii* (*The Journal of the American Medical Association*, April 7, 1951) discuss penicillin levels in the blood following the use of different types of penicillin. They state that when crystalline penicillin G in peanut oil and other oils was used with 4·8% of white wax, 300,000 units daily gave an average of 0·009 unit per millilitre, 600,000 units gave 0·055 unit per millilitre, and 1,000,000 units gave 0·133 unit per millilitre. If prolonged blood levels are required, 300,000 units every twelve hours must be given or 600,000 units every twenty-four hours. Crystalline penicillin G procaine in oil gave higher concentrations: 0·17 unit per millilitre with 300,000 units, 0·196 unit per millilitre with 600,000 units; 300,000 units daily gave a continuous blood level. For syphilis the authors gave 600,000 units on Monday and Wednesday and 900,000 units on Friday, producing continuous blood levels. Crystalline penicillin G procaine in oil containing 2% of aluminium monostearate gave the same results. Crystalline penicillin G procaine in oil containing 4% aluminium monostearate gave more prolonged penicillin blood levels: 1,200,000 units gave 0·4 unit per millilitre for twenty-four hours, 0·37 unit per millilitre for forty-eight hours and 0·014 unit per millilitre at the end of ten days. Crystalline penicillin G procaine for aqueous injection gave similar results to penicillin in oil and wax, penicillin G in oil, and penicillin in oil with 2% of monostearate and with 4% of monostearate. The aqueous solution, however, gave poor results after twenty-four hours. For syphilis the authors advise 3000 to 6000 units per pound of body weight. They state that the most prolonged effect was obtained when they used penicillin procaine with aluminium monostearate, penicillin procaine with 2% of monostearate, penicillin procaine in aqueous solution, and penicillin in oil and wax.

Amœbiasis.

J. A. BARGEN (*The Journal of the American Medical Association*, March 17, 1951) discusses present-day treatment of amœbiasis, from which 10% of Americans are said to suffer. He states that diagnosis is made by microscopic examination of a recently passed stool (within thirty minutes) for motile *Entamœba histolytica* and cysts. At least three loose stools may have to be examined. Complement-fixation tests are not reliable, though a positive result may be helpful in cases of suspected liver abscess. Proctoscopic examination is helpful in revealing the ulcers, which appear on prominent folds of the intestine, have undermined edges, a greyish white cap and a diameter of from two millimetres to three centimetres. X-ray examination of the colon usually yields negative results, though rarely it reveals a deformity involving almost all the large intestine. Metastases may occur in liver, lungs, brain, pericardium, pleura and spleen, and may cause no sign except an unexplained fever. In treatment, emetine, penicillin, streptomycin and sulphonamide have been praised. Aureomycin, however, in doses of 2·5 grammes per day has been advocated. The author states that excellent results have been obtained with chloroquine 0·3 gramme given twice daily for two days and then 0·3 gramme given daily for twelve to nineteen days. The standard treatment for severe attacks of amœbic colitis is one grain of emetine hydrochloride every twelve hours until six grains have been given. For moderate attacks up to four grains will suffice. This may be repeated in a week. A total dosage of up to twelve grains is not toxic. Carbarsone or phenarsone sulphoxylate 0·25 gramme three times a day until twelve capsules (three grammes) have been taken is given at the same time.

Pulmonary Tuberculosis.

S. J. SHANE *et alii* (*The Canadian Medical Association Journal*, April, 1951) describe the treatment of pulmonary tuberculosis with thiosemicarbarsones. Twenty-five patients were treated with 50 milligrammes daily in the first week, 100 milligrammes daily in the second week and 200 milligrammes daily for up to three months. The patients had moderately advanced or advanced tuberculosis and were not suited for surgery. The authors state that the results demonstrated the great toxicity of the preparation. Only 17 patients completed the course; toxic effects, spread of the disease or neutropenia prevented the others from finishing it. The toxic effects were nausea, vomiting, diarrhœa, headache, drug fever, chills, watering eyes, pruritus and enlarged lymph nodes. The principal observation made was that the drug was too toxic for general use.

Post-Traumatic Cerebral Syndromes.

J. CLIFFORD RICHARDSON (*The Canadian Medical Association Journal*, May, 1951) discusses post-traumatic cerebral syndromes and refers to the acute, convalescent, residual and delayed symptoms of head injury. The view is expressed that concussion causes prolonged disturbances in higher cerebral function as well as the initial unconsciousness, and the importance of detailed observation of post-traumatic confusional states is emphasized. The author states that with the great majority of head injuries the patients run their clinical course of concussion with or without focal contusion and recover within a period of several weeks. A small percentage of patients with severe brain injury recover slowly over a period of one to two years and are often left with residual mental impairment. Various focal signs of brain injury may persist. Severe intellectual loss is readily recognized, but milder degrees of impairment may be readily missed and misinterpreted. The later convalescent symptoms of subsiding brain injury may last for many months and may include giddiness, nervousness, headaches, and intolerance of alcohol. This post-concussive syndrome may be

identified by the characteristic symptoms and their remitting course. Various adverse factors may implant neurotic reactions during this recovery stage. Occasionally, personality change and a tendency to periodic headaches seem to persist on an organic basis. With the exception of those due to post-traumatic epilepsy and subdural hæmatoma, symptoms developing for the first time some weeks or months after the injury are usually psychoneurotic.

The Pathogenesis of Atherosclerosis.

J. LYMAN DUFF (*The Canadian Medical Association Journal*, May, 1951) discusses the pathogenesis of atherosclerosis and states that knowledge of this disease today is confined largely to what is known about the behaviour of cholesterol in the body. The deposition and accumulation of cholesterol in the intima of arteries has come to be regarded as the central feature of the disease. There exists a fairly satisfactory body of knowledge concerning the morphological characteristics of human atherosclerosis and its experimental counterpart, experimental cholesterol atherosclerosis. It is in regard to mechanisms or dynamics of development that we are particularly ignorant. Diets of low fat and low cholesterol content offer no real hope of lowering the blood cholesterol level significantly, though it is conceivable that they may alter the physico-chemical state of cholesterol in the blood without changing its quantity. Such diets may bring about clinical improvement through loss of weight or for other reasons, but there is no sure evidence to indicate that they offer a means of reversing the atherosclerotic process. The use of choline in the treatment of this disease has as its only basis the experimental studies that indicate a slight effect of choline in retarding the development of experimental cholesterol atherosclerosis under certain conditions. No clinical studies have been reported that establish convincingly any beneficial effect of choline in cases of atherosclerosis. At present, any form of treatment directed toward the reversal of the lesions in the arteries, even if such treatment should be conclusively demonstrated by well-controlled clinical tests to be of clinical benefit, must be regarded for the time being, at least, as purely empirical.

Sciatica.

C. H. MILLIKAN (*The Journal of the American Medical Association*, January 6, 1951) discusses sciatica. He states that lesions of nerve roots cause sciatica, and 90% of these are due to protruded intervertebral disks. Other causes, such as neoplasm, fracture, spondylolisthesis, osteomyelitis, syphilis, arthritis and epidural abscess, may cause the pains, and trauma, diabetes, alcohol and dietary deficiencies may cause neuritis. The pain extends from the buttock down the back of the thigh and leg, and even to the ankle and sole of the foot. Radiography and myelography may reveal narrowing of the space between the fourth and fifth lumbar vertebræ. Surgery may be necessary for prolonged or repeated sciatica. Operation is not indicated in a mild first attack of sciatica, when there is a large functional element, or when there are no physical signs, or when no pain exists. Surgery may fail in any case; pain may continue, and disk protrusion may recur. Treatment involves complete rest in bed for two or more weeks; the bed must be hard, and traction on a leg may help. Bathroom privileges and getting up for meals may be allowed. The patient may be very uncomfortable, and great encouragement may be necessary to maintain bed rest. Administration of aspirin and codeine may be of assistance. A corset may help to support the lumbar region, though the effect may be mainly psychological. Correct lifting and the avoidance of sudden twists and strains of the back should be advised. Operation on a disk should be performed only when the patient has tried all other treatment carefully, and has come to the end of his tether. In any case neurotic patients should not be operated upon, and first attacks and absence of pain are contra-indications.

Treatment of Neoplasms.

D. A. KARNOFSKY *et alii* (*A.M.A. Archives of Internal Medicine*, April, 1951) report on the effect of triethylene melamine in the treatment of neoplastic disease. They state that the substance is akin to nitrogen mustard. Its intravenous injection produced transient improvement in patients with Hodgkin's disease, lymphosarcoma, and chronic lymphatic and myelogenous leuchæmia. A dosage of two to three milligrammes was given daily up to a total of three to twenty milligrammes. The drug rarely caused vomiting or thrombosis at the site of injection, as nitrogen mustard did. Oral administration in a dosage of 20 to 40 milligrammes in five weeks caused nausea, but produced temporary improvement in patients with the same diseases. With carcinoma results were unsatisfactory. The drug is only a palliative.

Serum Cholinesterase.

L. J. VORHAUS *et alii* (*The American Journal of the Medical Sciences*, February, 1951) discuss the value of the serum cholinesterase test in acute hepatitis. Seven patients with acute infectious hepatitis, homologous serum jaundice and acute toxic hepatitis were tested throughout the course of illnesses lasting three to ten weeks by various liver function tests (ten in all). The authors found that serum cholinesterase tests gave a more exact picture of the condition of the liver (the hepato-cellular activity) than any of the other ten tests—serum albumin and globulin and bilirubin determinations, thymol turbidity and flocculation tests, cephalin bromsulphalein retention, prothrombin time estimation, and urine urobilinogen, serum alkaline phosphatase and serum cholesterol determinations. None of these other tests measured the liver activity so well.

Staphylococcus Bacteriæmia.

S. C. KRAVITZ AND C. N. BREED (*The Journal of the American Medical Association*, March 17, 1951) report a case of *Staphylococcus aureus* bacteriæmia cured by antibiotics and surgery. They quote a statement that the death rate has been reduced from 85% to 20% by penicillin, and state that abscesses, endocarditis and generalized bacteriæmia caused a high mortality. The treatment consisted of heavy dosage of effective antibiotics early in the disease, continuation till the disease was eradicated and evacuation of pus. In the authors' case there were prolonged fever, operation on an echinococcus cyst and subsequent septicæmia. After operation 300,000 units of penicillin were given twice daily, and later 200,000 units of sodium penicillin every three hours and 0·5 gramme of streptomycin every four hours. Rigors and high fever persisted, and blood culture yielded a growth of *Staphylococcus aureus*. Ten million units of penicillin were given daily, with 500 milligrammes of aureomycin every six hours and 0·5 gramme of dihydrostreptomycin intramuscularly every six hours. Blood culture ceased to yield results. The patient remained seriously ill with cough and chest pain without fever. Eventually operation revealed a golden yellow spongy mass which represented an absorbable gelatin sponge left at a previous operation. Its removal was followed by dramatic improvement. The antibiotic therapy was stopped ten to eighteen days after operation, and the patient made a rapid recovery.

Psychomotor Epilepsy.

F. M. FORSTER (*The Journal of the American Medical Association*, January 27, 1951) discusses therapy for psychomotor epilepsy. He states that the attacks occur in late childhood or adult life. They are known also as epileptic equivalents and epileptic fugue states. In the attacks there occurs psychic or motor activity irrelevant for the time and place. The attack lasts one to five minutes, and frequency varies greatly. Amnesia follows. Smacking of the lips or uncinate symptoms may occur. In the diagnosis major epilepsy and *petit mal* must be excluded. Electro-encephalograms are said to be helpful; these have shown that psychomotor seizures may be manifestations of focal epilepsy. It is said that phenacetylurea is effective. "Dilantin", phenobarbital, mesantoin, and trimethyloxazolidine dione were used with fair success. Diphenylhydantoin sodium in daily doses of 0·3 to 0·5 gramme was mainly employed prior to the use of phenacetylurea. The last 27 of the author's patients have been treated with phenacetylurea, 1·5 to 3·5 grammes per day; 13 were cured, but four became so depressed that the drug had to be remitted. Temporal lobe lesions were found in refractory cases, and operation on the temporal lobe with surgical excision of a part of the lobe was beneficial in several cases. Liver and blood disorders may occur with phenacetylurea, and the authors state that "Dilantin" and phenobarbital are often satisfactory and perhaps less toxic.

Liver Function Test.

J. W. NORCROSS *et alii* (*The American Journal of the Medical Sciences*, February, 1951) discuss the bromsulphalein liver function test, with special reference to renal excretion. They state that five milligrammes of bromsulphalein per kilogram were injected into a group of healthy nurses and physicians. The amount of the dye excreted in the urine was an average of 1·2% of the amount injected. This they consider so small an amount as to be negligible. They state that the test depends on the fact that the dye is excreted almost entirely by the Kupffer cells of the liver. When the liver is damaged excretion of the dye is diminished. The investigation is stated to show that renal excretion does not affect the validity of the test of liver function.

British Medical Association News.

SCIENTIFIC.

A MEETING of the New South Wales Branch of the British Medical Association was held at the Royal North Shore Hospital of Sydney, Crow's Nest, New South Wales, on June 21, 1951. The meeting took the form of a series of clinical demonstrations by members of the medical and surgical staff of the hospital.

Porphyria.

DR. I. A. BRODZIAK presented a woman, aged thirty years, who had come under the notice of the hospital in December, 1950. At that time her history was one of generalized lethargy and lack of interest extending over a period of two years. In the past she had been admitted to the Reception House on five different occasions for attempted suicide, and once to another hospital with what was considered to be encephalitis. On her admission to the Royal North Shore Hospital of Sydney the patient had been mentally confused and delirious, and complained of generalized abdominal pain, nausea, vomiting and constipation. The constipation had been present for three weeks. On examination the patient was found to be thin and wasted, with generalized muscle weakness and hypoactive reflexes. She complained of abdominal tenderness, but there was no muscle guarding or rigidity. Her pulse rate was 120 per minute, and her blood pressure was 140 millimetres of mercury, systolic, and 110 millimetres, diastolic. No other abnormal physical signs were detected. However, her urine on one occasion was found to be darker in colour than normal, and the tentative diagnosis of porphyria was confirmed by the finding of porphyrins in abnormal concentrations in a twenty-four-hour specimen of urine. Dr. Brodziak presented details of the porphyrins found in a series of twenty-four-hour specimens of urine. He said that since the time of the first finding of porphyrins in the patient's urine, her symptoms of delirium, mental confusion, generalized abdominal pain and constipation had necessitated her readmission to the hospital on three occasions. Each attack, similar in pattern, had been observed to cease spontaneously after one week in hospital.

Agranulocytosis and Suspected Cerebral Abscess.

DR. F. A. E. LAWES presented a woman, aged thirty-seven years, who had first come under medical care at Bega in September, 1950, when she was diagnosed as suffering from an upper respiratory tract infection. A month later she had sinusitis, and pus was obtained on antral puncture. Her condition responded to treatment. About four weeks later she was again examined in Bega, and was found to be very ill, with a temperature of 103° F. Her condition did not respond to penicillin or sulphonamides, but responded very well to aureomycin. Her left frontal sinus was opened externally. She was first admitted to the Royal North Shore Hospital of Sydney in January, 1951, when she said that she had noticed various painful swellings, especially on the limbs and buttocks. She had a mild degree of pyrexia. The result of attempted blood culture at this stage was negative, and no pus was aspirated from the painful swelling in her thigh. A diagnosis of pyæmic abscesses was made. Blood examination revealed a hæmoglobin value of 12 grammes *per centum*, and a total leucocyte count of 13,300 per cubic millimetre, made up of neutrophile cells 71%, lymphocytes 19%, eosinophile cells 2%, monocytes 5% and band forms 3%. The patient was discharged from hospital early in February, 1951, but was readmitted on May 5 with the story that one week before her admission she had begun to vomit. The vomiting was forceful and recurred several times daily. Six days before her readmission to hospital she had begun to suffer from frontal headaches, which were present all the time, but varied in intensity. After several attacks of vomiting she had noticed small spots on her face. These were found to be petechial. She said that she had taken about 24 A.P.C. powders over this period for the headaches. For a few days before the onset of her illness she had noticed some diminution of her power of concentration. The results of examination of her nervous system were negative in all respects, except that her pupils were rather dilated and reacted sluggishly to light. Her ocular fundi were normal, and all other systems were normal. Lumbar puncture was performed, and the pressure was found to be 58 millimetres of cerebro-spinal fluid. Routine blood examination revealed a total leucocyte count of 5300 per cubic millimetre, made up of neutrophile cells 1%, lymphocytes 92% and monocytes 7%. She was immediately given penicillin. On the next day, May 8, she was very ill, was vomiting considerably and was becoming dehydrated. One litre of blood was given, and the administration of "Pentnucleotide", 20 millilitres twice a day, was commenced. On May 9 her leucocytes numbered 4800 per cubic millimetre, 3% being neutrophile cells; the platelets numbered 181,280 per cubic millimetre. On May 10 the number of leucocytes had fallen still further to 3800 per cubic millimetre, but the proportion of neutrophile cells had risen to 13%. By May 21 the total leucocyte count was down to 2400 per cubic millimetre, but the proportion of neutrophile cells had risen to 34%. Her condition had gradually improved, her headaches had diminished and her vomiting had ceased. Her temperature remained normal until May 20, when it rose to 101° F., and the next day it again rose to 101·4° F. It fell to normal temporarily, but on May 23 again rose to 102·4° F. She had no faucial ulceration, skin hæmorrhages, lymph gland enlargement or palpable spleen. On May 24 sternal puncture revealed a mild degree of maturation of the granulocyte series. The X-ray appearance of her skull and chest was normal. An attempt at air encephalography resulted in surface filling only. An electroencephalogram prepared at Sydney Hospital on May 17 was reported as being within normal limits. Her latest leucocyte count was 5000 per cubic millimetre, of which 56% were neutrophile cells.

Hypertension and Anxiety State.

Dr. Lawes then presented a woman, aged forty-seven years, who had been admitted to hospital on May 4, 1951, complaining of inability to walk straight since November, 1950, pain in the neck for twelve months and headaches for six years. She had been found to have hypertension when her headaches had first begun, and for that sympathectomy had been performed in October, 1950. She said that she had been well for a period of weeks after the operation, but had then developed inability to walk straight. She also suffered from palpitation and substernal pain, with an onset at rest and lasting a few minutes. She noticed spots before her eyes and her vision was blurred at times. Her memory was deteriorating. Examination of the patient revealed that she had a blood pressure of 245 millimetres of mercury, systolic, and 145 millimetres, diastolic. Her apex beat was in the sixth left intercostal space, four and a half inches from the mid-line. Examination of her ocular fundi revealed increased light reflex from the arteries, and nipping at the arterio-venous crossings. There were no other relevant findings from examination, except that the patient was very anxious and worried about herself. She was given a course of "Vegolysin"; for sixteen days her blood pressure values ranged about between 190 and 240 millimetres of mercury, systolic, and the diastolic value rose from 110 to 140 millimetres of mercury. On the twenty-first day the blood pressure fell to 150 millimetres of mercury, systolic, and 100 millimetres, diastolic.

Chronic Nephritis Complicating Pregnancy.

DR. F. H. HALES WILSON showed a married woman, aged twenty-one years, who had attended the pre-maternity clinic on August 17, 1950, when her blood pressure was found to be 140 millimetres of mercury, systolic, and 90 millimetres, diastolic, and her urine was normal according to routine tests. A month later her blood pressure was 110 millimetres of mercury, systolic, and 68 millimetres, diastolic, and there was a trace of albumin in her urine. On March 2, 1951, her blood pressure was 124 millimetres of mercury, systolic, and 80 millimetres, diastolic. No excessive increase in weight had occurred. Her urine contained a cloud of albumin, and microscopic examination revealed a few pus cells, red blood cells and granular casts. On March 17 she was admitted to hospital with a blood pressure of 140 millimetres of mercury, systolic, and 98 millimetres, diastolic, and a blood urea content of 56 milligrammes *per centum*. By March 29 the blood urea value had risen to 89 milligrammes *per centum*, and the blood pressure to 150 millimetres of mercury, systolic, and 110 millimetres, diastolic. On March 30 induction of labour was carried out at the thirty-eighth week of pregnancy The blood pressure was 140 millimetres of mercury, systolic, and 110 millimetres, diastolic. The labour lasted for six hours twenty minutes, and a female infant weighing five pounds nine and three-quarter ounces was born. On April 9 the hæmoglobin value of the patient's blood was 6·6 grammes *per centum*, so one litre of blood was given as a transfusion. On April 11 microscopic examination of the urine revealed only occasional red blood cells. The blood pressure was 150 millimetres of mercury, systolic, and 100 millimetres, diastolic. The blood urea content was 132 milligrammes *per centum*. The urea clearance was 10% of the average normal value. The blood carbon dioxide combining power was 32 volumes *per centum* of

carbon dioxide, as compared with normal values of 54 to 72 volumes *per centum* of carbon dioxide. The serum calcium content was within normal limits, being 9·4 milligrammes *per centum*. The serum phosphorus content was 6·5 milligrammes *per centum*, a value above the normal range of two to five milligrammes *per centum* calculated as inorganic phosphate. The minimum urine specific gravity value noted while the patient was in hospital was 1004, and the maximum value was 1016. The patient was discharged from hospital on April 23. Her blood pressure then was 120 millimetres of mercury, systolic, and 80 millimetres, diastolic, and the urine contained a faint cloud of albumin.

Dr. Hales Wilson commented that the case was of special interest in that successful pregnancy had occurred in the presence of severe renal impairment. The absence of hypertension was probably the reason. He had shown the patient by courtesy of Dr. Edmund Collins, her honorary obstetrician.

Coarctation of the Aorta and Pregnancy.

Dr. Hales Wilson's second patient was a married woman, aged twenty-three years, who at her first visit to the pre-maternity clinic in June, 1950, had been noted to have a blood pressure of 200 millimetres of mercury, systolic, and 66 millimetres, diastolic. Classical clinical signs had suggested, and X-ray examination had confirmed, the diagnosis of coarctation of the aorta. When confined at term, the patient had a blood pressure of 220 millimetres of mercury, systolic, and 110 millimetres, diastolic, and no albuminuria. Labour lasted for eight hours twenty-five minutes, being assisted by pudendal block and instrumental delivery, and resulted in the birth of a living female infant weighing seven pounds five ounces. There was no evidence of cardiac decompensation at any time. The puerperium was uneventful. Dr. Hales Wilson said that he was showing the patient by courtesy of Dr. A. A. Moon, her honorary obstetrician.

(*To be continued.*)

Out of the Past.

In this column will be published from time to time extracts, taken from medical journals, newspapers, official and historical records, diaries and so on, dealing with events connected with the early medical history of Australia.

GOVERNOR PHILLIP TO LORD SYDNEY.[1]

[EXTRACT.]

12 Feb., 1790.

Whether the smallpox, which has proved fatal to great numbers of the natives, is a disorder to which they were subject before any Europeans visited this country or whether it was brought by the French ships, we have not yet attained sufficient knowledge of the language to determine. It never appeared on board any of the ships in our passage, nor in the settlement, until some time after numbers of the natives had been seen dead with the disorder in different parts of the harbour, and two men with a boy of about eight years of age and a girl of eleven, had been brought to the hospital, in the smallpox.

Both the men died, but the boy and girl recovered. These people were brought up the middle and the latter end of April, and the smallpox never appeared in the settlement until the 2nd of May, when a man belonging to the Supply was seized with the disorder and died a few days afterwards: nor has it ever appeared in the settlement except on that man, and the native who caught the disorder from the children.

Correspondence.

PRE-BRONCHIECTASIS.

SIR: In the journal of August 25 Mr. H. B. Harwood makes a strong plea for the early recognition and treatment of what he calls "pre-bronchiectasis". No one would question the truth of the majority of his statements, but many would deny the prominence he accords to bronchoscopy as a method of treatment.

[1] From the original in the Mitchell Library, Sydney.

It is stated that "the reversibility of bronchiectasis in the early stages is now an established fact". This is undoubtedly true in that patients with cough and sputum and demonstrable dilatation of some of the bronchi have been observed to return to normal both clinically and bronchographically. However, it should be noted that many such patients have done so without ever having been subject to bronchoscopy.

Even allowing that the term bronchiectasis covers a number of conditions, both clinical and pathological, I do not believe that anyone really knows whether the type of case quoted by Mr. Harwood does develop established bronchiectasis, and therefore would question the use of the term "pre-bronchiectasis".

The most enthusiastic bronchoscopist can aspirate material only from the main or lobar bronchi. The orifices of the subdivisions of the lobar bronchi are only a few millimetres in diameter, and each rapidly subdivides up to twenty generations to terminate in the alveoli. To suggest that such a branching system of fine capillary-like tubes, closed peripherally, can either be sucked out or washed out is absurd. Clearing of any but the main or lobar bronchi can be done only by the action of coughing and/or the action of cilia, and the virtue of bronchoscopy is in the coughing it induces.

Mr. Harwood suggests that patients who have a persistent cough eight or ten weeks after a serious illness such as whooping-cough should be investigated, and if necessary, receive active treatment. He states that this should be "to establish a clear and adequate airway to the alveoli, and to reestablish normal physiological upward drainage by ciliary wafting, tussive squeeze and the blast of the cough itself". The reestablishment of normal physiology is best accomplished by the physiological methods of physiotherapy which the patient can apply each day, and not by the unphysiological act of bronchoscopy applied once a month, once a fortnight or even once a week. It must be admitted that it is difficult to get the cooperation of some children under the age of five years, but this is a matter of personality, perseverance and skill on the part of the physiotherapist. Children of tender years should attend a "class" where their capacity for quick imitation of older children can be utilized. The final point I would emphasize is that chest physiotherapy, to be effective, can be entrusted only to someone competent and experienced in this work.

Yours, etc.,
IAN MONK.

135 Macquarie Street,
Sydney,
September 3, 1951.

THE NORTHCOTT NEUROLOGICAL CENTRE.

SIR: The Northcott Neurological Centre, which is owned and controlled by the Returned Sailors', Soldiers' and Airmen's Imperial League of Australia, New South Wales Branch, will be available for the reception of patients about October 1 next. I should like to make the following information concerning the aims and working methods of the centre available to members of the British Medical Association.

The Returned Sailors', Soldiers' and Airmen's Imperial League of Australia has purchased the hospital "Glengarlen" in Lytton Street, North Sydney, to house the centre, and this building has now been completely remodelled and equipped with all the diagnostic machinery necessary for neurological investigation. Dr. George Selby, M.B., M.R.C.P., has been employed full time as the centre medical officer, and a fully trained nursing and technical staff appointed. The Returned Sailors', Soldiers' and Airmen's Imperial League of Australia has selected a group of 25 consultants, including neurologists, psychiatrists, neurosurgeons and a number of specialists in related fields.

The function of the centre is essentially diagnostic, and no treatment, either out-patient or in-patient, is contemplated other than in a limited number of special or urgent cases. A few in-patient beds have been provided to deal with admission and discharge cases and such special procedures as arteriography, myelography and air studies.

Admission to the centre may proceed only through two channels: (i) By reference from an officer appointed by the Returned Sailors', Soldiers' and Airmen's Imperial League of Australia at headquarters (273 Elizabeth Street, Sydney) for this purpose. (ii) By direct reference by letter from the patient's own doctor. In this case the patient will be referred back to his doctor with the results of investigations and recommendation for treatment, unless the referring doctor specifically requests that the centre arranges for disposal of the patient for treatment. Where possible it is intended to see patients by appointment, and they

should be informed, therefore, that it is necessary for them to ring the centre, stating that they have a letter of reference from their own doctor, and arrange an appointment. When referring a case by letter, the patient's doctor is asked to state that the patient is an ex-serviceman or the dependent relative of an ex-serviceman.

The aim of the centre is to provide facilities for immediate neurological, psychiatric and neurosurgical investigation for ex-servicemen and women and their dependent children and relatives.

The State Council of the Returned Sailors', Soldiers' and Airmen's Imperial League of Australia would be grateful if this letter could be published in THE MEDICAL JOURNAL OF AUSTRALIA.

Yours, etc.,
GILBERT PHILLIPS,
Honorary Consultant Director.

Sydney,
August 31, 1951.

POST-GRADUATE STUDY IN THE UNITED STATES OF AMERICA.

SIR: Many of our best athletes and swimmers have gone to the United States of America to study at institutions. Surely it would be better for Australia if some of our best young medical graduates were given scholarships to study in the United States of America. Imagine what twelve months at the Mayo Clinic and twelve months at the Johns Hopkins Hospital would do for a young surgeon, or similar periods with Lempert in New York and Chevalier Jackson in Philadelphia for an otolaryngologist.

Yours, etc.,
F. W. SIMPSON, D.O. (Oxon.).

105 St. George's Terrace,
Perth,
August 17, 1951.

Obituary.

JAMES ALFRED LARWILL.

WE are indebted to Professor S. Sunderland for the following appreciation of the late Dr. James Alfred Larwill.

The sudden death of Dr. James Alfred Larwill on April 5 came as a great shock to a wide circle of friends and associates. Though he had suffered a severe cardiac illness in 1944 which permanently damaged his heart, few knew of it, and from his cheerful bearing none could have guessed it. His death came unexpectedly to many and to the deep regret of all.

Educated at Brisbane Grammar School, Fred Larwill did not commence his medical course until after the first World War, when he entered the Melbourne medical school as a young ex-serviceman who had served overseas as a pilot with the Australian Flying Corps with the rank of lieutenant in the Second Flying Squadron. In 1925 he graduated M.B., B.S. with honours in all final subjects (medicine, surgery, and obstetrics and gynæcology), and a high place on the final list ensured his appointment to the resident medical staff of the Melbourne Hospital, where he remained for twelve months. After this appointment he entered private practice in Moreland. He rapidly established himself in the community not only as a sound and skilful practitioner, but also as a wise and understanding confidant. This period of his career was passed with conspicuous success. Despite the exacting demands of a large and busy practice, Larwill managed to find time to maintain a close association with the medical school and his old hospital. In the former he served for many years as a part-time demonstrator in histology, and on one occasion as acting lecturer, and in the latter as an honorary clinical assistant. He was interested in all outdoor sports, particularly in bowls and football, being for several years honorary medical officer to the Melbourne Football Club.

Driven by a strong sense of duty and responsibility to his patients, pressure of work, particularly over the war years, took heavy toll of his health, and in 1944 he suffered the severe illness which terminated his career as a general practitioner. At this time there was a vacancy on the staff of the anatomy department for a senior lecturer in histology and embryology. Dr. Larwill's long association with the department as a part-time officer responsible for instruction in these subjects, together with his numerous other qualities, made him the obvious person to fill this vacancy if his health would permit him to accept the post. Fortunately this became possible. Though he was no stranger to the department, we were delighted to welcome him as a full-time colleague. He entered on his new duties with rare enthusiasm and took a lively interest in all departmental activities. He was a clear and attractive lecturer with a personality that awakened not only interest and respect but also affection. There is abundant testimony that his reputation stood high with his students.

But Dr. Larwill was more than a good instructor. He was the ideal colleague, and those of us who knew him well will always think of him as a loyal and lovable friend. He came to the department at a critical period, when attempts were being made to rebuild the staff which the war years

had eroded away. None could resist his sincerity, essential friendliness and desire to help, and these qualities did much to make new members feel at home and to create the happy atmosphere which prevails in the department. Years in general practice had given him a wide and deep knowledge of men and human affairs, and he was quick to sense the difficulties and problems of others. We can speak only in the most grateful terms of the valuable assistance that he rendered the department in a variety of ways and of the unsparing help and encouragement that he gave others at every opportunity, more especially the younger generation for whom he had a fond attachment. We deeply regret that he was not permitted to remain longer with us as a colleague and a friend. He is survived by his widow, two sons and a daughter, to whom we extend our sincere sympathy.

HERBERT HENRY ERNEST RUSSELL.

WE are indebted to Sir Henry Newland for the following appreciation of the late Dr. Herbert Henry Ernest Russell.

Colonel Herbert Henry Ernest Russell, O.B.E., M.D. (Adelaide), M.B. (Melbourne), F.R.C.P. (Edinburgh), who died on July 10, 1951, was one of those men who, without adventitious aid, but by sheer merit, climb unobtrusively to high positions in the service of their country.

When we were medical students at the University of Adelaide, membership of the Lacrosse Club brought us together for the first time. The friendship made then endured for over fifty years. Bert Russell, as he has always been known to all who knew him, believed that work, as Osler has said, was the master key to success. In his steadfast adherence to it, ever a shy man, he hid his light under a bushel.

He was born in Adelaide on October 28, 1875, a son of the late J. T. Russell, of Unley, South Australia. He was educated at Prince Alfred College, Adelaide, and at the Universities of Melbourne and Adelaide. This dual relation arose from the enforced migration of Adelaide medical students at the time of the "hospital row" in the nineties of last century. After graduating at the University of Melbourne he gained the doctorate degree at the University of Adelaide. Proceeding overseas, he later on passed the examination for the membership of the Royal College of Physicians of Edinburgh, and was in due course elected a Fellow of the Royal College of Physicians of England. He settled in Unley and soon acquired a large practice, which he held until the date of his death. The claims on private practice, incessant as they were, he never permitted to interfere with the conscientious adherence to the duties of medical officer of health for the City of Unley, a position Russell held for forty-five years.

Fired by the desire to serve his country, he joined the Commonwealth Military Forces in 1905, and rose to be Principal Medical Officer, Fourth Military District, during the greater part of World War I. In those strenuous days he organized the first Voluntary Aid Detachments. He also established "curative workshops" for the badly wounded returned soldiers. This led to "vocational training", which was the forerunner of the present "occupational therapy". For this fine service he received the O.B.E.

From 1928 onwards he was State Controller of Voluntary Aid Detachments, raising over twenty detachments during World War II. The only detachment raised since then is the existing Number 400 "Russell" Voluntary Aid Detachment.

Russell's service to the Red Cross was also of a high order. A member of the Divisional Council and Executive Committee, he was also chairman of the Medical Advisory Committee (his own creation). He was also chairman of the Convalescent Homes Committee and of the Emergency Services Committee. As Director of Emergency Services he was responsible for the establishment of volunteer service detachments and transport section, in which formations 2000 women were fully trained by September, 1939. For his valuable and consistent service to the Red Cross Society he was awarded its greatest honour—honorary life membership.

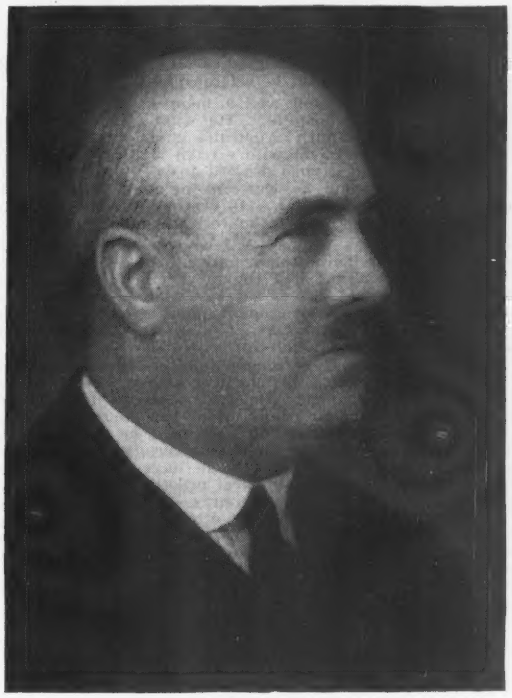
Ever since 1909 when he became an honorary lecturer and examiner to Saint John Ambulance Association, Russell remained closely associated with the activities of the Venerable Order of the Hospital of Saint John of Jerusalem. He was awarded the service medal and bar. Promotion from Serving Brother (1935) to Officer and Commander culminated in his creation as Knight of Grace in 1947, and in his membership of Chapter of Priory within the Commonwealth of Australia. In 1950 he became an honorary life member of the Saint John Ambulance Association.

Russell was at various stages in his long career associated with hospitals and nursing bodies. His positions included honorary anæsthetist to the Adelaide Children's Hospital, honorary assistant bacteriologist to the Adelaide Hospital (1904), honorary obstetrician and member of the committee of management of Queen's Home, honorary physician to Minda Home (1908-1932), and later honorary consulting physician, a member of the Metropolitan Infectious Diseases Hospital board of management from its inception in 1932, and chairman of its medical and house committee. He became a member of the Nurses' Board in 1925, and has been an examiner in midwifery and care and feeding of infants for the Nurses' Registration Board. He also held the office of chairman of the South Australian Branch of the Royal British Nurses' Association from 1925.

Russell held many of the offices of the South Australian Branch of the British Medical Association. He served on the Branch Council for several sessions, and was a past president. After fifty years' membership he became an honorary life member in 1949. His public health interests led to membership of the Examination Board for South Australia (honorary) of the Royal Sanitary Institute of London for twenty-two years. For many years prior to his death Russell was on the directorate of the British Medical Hall Company, a body formed for the purpose of providing a house for the South Australian Branch of the British Medical Association. As chairman of the board I can testify that the recent acquisition of a splendid site in North Adelaide overlooking the city gave Russell great satisfaction. Other interests of his were a pastoral property and ownership of a racehorse. Just how profitable these were I am not in a position to say. Wedded to his profession, he never married.

H. H. E. Russell was what he was proved to be, a general practitioner, and a good one at that. In partnership with his younger and devoted brother, E. A. H. Russell, he had a very large practice in Unley. He was modest, tactful and a good administrator. He had a good sense of humour and an explosive laugh. It is certain that he will be greatly missed in Unley, where he had resided and practised for over half a century.

Sir George Wilson writes: As a very old friend and one who was associated with him for many years, I would like to add my word of appreciation to the life and work of Dr. H. H. E. Russell—"Bert Russell" as he was to his friends. By his death Adelaide has lost one of its best known and well liked general practitioners, and one who had always been to the front in any work for the community at large during the last fifty years. It is more particularly to Russell's interest in the practice of obstetrics that I would like to refer. When Russell, then aged twenty-four years, took his M.R.C.P. in Edinburgh, his work had so impressed Milne Murray that he presented Russell with a pair of his original obstetrical forceps, which were to be useful to him in later life and which he always treasured. For many



years in his general practice at Unley he was doing a tremendous amount of midwifery—as many as 400 cases in one year; and this was in the days when midwifery patients were attended mostly in their own homes, very often with only the assistance of the local "Sairey Gamp". Nothing pleased Russell more than to take a resident medical officer or a senior medical student with him when he went to attend an extern midwifery patient in his district for the old Benevolent Obstetrical Hospital and to demonstrate to them how by improvising and using whatever was available it was possible to manage and conduct the ordinary case of labour even in the poorest home. Many resident medical officers and students must have gained valuable experience from these cases.

For a great many years he did practically all the midwifery work at the Fullarton Refuge, which at that time worked largely in conjunction with what was in those days the Queen's Home, but is now the Queen Victoria Maternity Hospital, and the main teaching hospital for medical students and nurses in South Australia. Russell was appointed to the honorary staff of this hospital in 1910 and gave his services until 1928, when he was appointed to the consulting staff. He always took a keen interest in the work of the hospital up till the time of his death. During his time on the active staff of the hospital it was the custom to have periodical meetings of the honorary staff to discuss various obstetrical problems, and I well remember how on many occasions Russell consistently advocated rupturing of the membranes in cases of impending eclampsia.

This was before the days when taking of the blood pressure was the routine in ante-natal cases. Russell's test was dependent on whether the amount of albumin in the urine was increasing. He would test the urine for albumin four-hourly, and if the albumin was increasing he looked on this as a definite indication to separate the membranes from the lower uterine segment and rupture the bag of membranes. His belief was that the eclamptic condition was due to increased pressure in the lower uterine segment. He consistently taught and practised this for over forty years—long before any such treatment was advocated for these cases—and his results, as compared with the unfavourable results associated with developed eclampsia in those days, were remarkably good. His great point was not to let the patient have a fit. He admitted that in those rare cases in which there was no albuminuria, the condition could not be anticipated, but in the ordinary case the progressive increase of albumin was the definite indication to get labour started. Looking back to those days, when often a patient would be admitted to hospital with a history of having had from 12 to 20 fits, makes one realize what a difference the routine ante-natal supervision has made in midwifery practice. Though Russell wrote very little in the journals, to those who knew his work he must be looked on as one of the pioneers in the prevention of eclampsia.

For some years Russell was a member of the board of management of the hospital. He was a member of the Nurses' Board of South Australia and was the official examiner for nurses in midwifery from 1923 up till the time of his death. I had a good deal to do with Russell and his work during the time he was on the staff of the Queen Victoria Maternity Hospital. He was a loyal colleague and those of us who knew him intimately will miss him sadly.

Dr. F. J. Douglas writes: A few days ago I read with sorrow Bert Russell's obituary notice. As a fellow student in those far-off days I had the privilege of knowing Bert intimately and would like to pay my tribute to his memory. He was a splendid type of student, honest and hard working at work or play, and was very popular with his fellows. He was one of the first batch of students who, owing to a dispute between the Government and the Adelaide Hospital Board, had to complete his medical course elsewhere, and he qualified in Melbourne. In sport he excelled at lacrosse, which in his student days was the most important university winter game, and in Melbourne he continued that interest and was a member of the university team that won the premiership. Since our student days we had followed different paths—Bert as a general practitioner with a large city practice and I in the country—but we were always great friends even if we did not often meet. I have never heard anyone say an unkind word about Bert, and he never spared himself. He was loved and respected by his patients, and the world is the better for Bert Russell's life, and the poorer through his death. My sincere sympathy is with his family.

EDMUND WESLEY FINKLE.

We regret to announce the death of Dr. Edmund Wesley Finkle, which occurred on September 11, 1951, at Perth.

WILLIAM AMBROSE SPRING.

We regret to announce the death of Dr. William Ambrose Spring, which occurred on September 8, 1951, at Ballarat, Victoria.

Post-Graduate Work.

THE POST-GRADUATE COMMITTEE IN MEDICINE IN THE UNIVERSITY OF SYDNEY.

Course in Occupational Medicine.

The Post-Graduate Committee in Medicine in the University of Sydney wishes to announce that a course in occupational medicine for general practitioners will be held on Wednesday afternoons from September 26 to November 21, 1951, under the supervision of Dr. G. C. Smith. The fee for the course will be £1 1s., and those wishing to enrol should communicate with the Secretary, the Post-Graduate Committee in Medicine, 131 Macquarie Street, Sydney, telephone numbers BU 5238, BW 7483. The programme will be as follows:

September 26, 1951, at the School of Public Health and Tropical Medicine, University of Sydney: 2 p.m., "Introductory: The Scope of Occupational Medicine. Facilities and Services Available. Statutory Requirements", Professor E. Ford and Dr. G. C. Smith; 3.30 p.m., "The Relationship between General Medical Practice and Industrial Medical Practice", Dr. J. C. G. Hadley.

October 3, 1951: 2 p.m., visit to Australian Paper Manufacturers, Limited, McCauley Street, Matraville, Dr. J. C. G. Hadley.

October 10, 1951, at the School of Public Health and Tropical Medicine, University of Sydney: 2 p.m., "The Relationship between Occupation and (*a*) Cardiac Disease and (*b*) Pulmonary Tuberculosis", Dr. W. T. Nelson; 3.30 p.m., "Dust in Industry", Dr. G. C. Smith.

October 17, 1951: 2 p.m., visit to Taubmans, Limited, Mary Street, St. Peters, Dr. W. T. Nelson.

October 24, 1951, at the School of Public Health and Tropical Medicine, University of Sydney: 2 p.m., "Poisoning by Metals", by Dr. G. C. Smith; 3.30 p.m., "Diagnosis of Common Skin Conditions which Might be Confused with Those of Occupational Origin", Dr. F. C. Thompson.

October 31, 1951: 2 p.m., visit to Chrome Chemicals (Australia) Proprietary, Limited, Grand Avenue, Parramatta, Dr. J. H. Blakemore.

November 7, 1951, at the School of Public Health and Tropical Medicine, University of Sydney: 2 p.m., "Poisoning by Fumes and Gases", by Dr. G. C. Smith; 3.30 p.m., "Vision Standards and Industrial Eye Injuries", Dr. H. M. Taylor.

November 14, 1951: 2 p.m., visit to Railway Workshops, Dr. M. R. Finlayson and Dr. A. Callen.

November 21, 1951, at the School of Public Health and Tropical Medicine, University of Sydney: 2 p.m., "Common Industrial Injuries", Dr. E. M. Sheppard; 3.30 p.m., "Rehabilitation and Placement of the Physically Handicapped Worker", Dr. M. R. Finlayson.

Royal Australasian College of Surgeons.

NOTICE.

The Honorary Secretary of the New South Wales State Committee of the Royal Australasian College of Surgeons announces that a meeting will be held in the Stawell Hall, 145 Macquarie Street, Sydney, on Wednesday, October 10, 1951, at 8.15 o'clock p.m. The subject for discussion will be "Facial Neuralgias". Papers will be read by Mr. R. A. Money and Mr. D. G. Carruthers; the discussion will be opened by Dr. Eric Susman. All members of the medical profession are invited to attend.

The Royal Australasian College of Physicians.

ALLOCATIONS OF CORTISONE.

Medical practitioners are reminded that The Royal Australasian College of Physicians is responsible for the allocation of cortisone throughout the Commonwealth and will consider applications for the use of the hormone in the treatment of (*a*) acute rheumatic fever with carditis, (*b*) rheumatoid arthritis with florid and reversible manifestations, particularly for patients in the younger age and wage-earning groups, (*c*) Still's disease, (*d*) Addison's disease, (*e*) *status asthmaticus*, if life is endangered, (*f*) acute disseminated *lupus erythematosus*, (*g*) exfoliative dermatitis (severe), (*h*) drug reaction, if life is endangered, (*i*) pemphigus (severe), if life is endangered, (*j*) acute inflammatory eye diseases, if sight is endangered, (*k*) very severe burns and/or shock, if life is endangered, (*l*) other medical or surgical emergencies in which the use of cortisone would be a life-saving measure.

Applications should include all relevant clinical details and laboratory findings and should be addressed to the Honorary Secretary, The Royal Australasian College of Physicians, 145 Macquarie Street, Sydney.

It is emphasized that applications should be made to the State subcommittees appointed by the College only when cortisone is urgently required in cases of acute emergency or when life is endangered.

Naval, Military and Air Force.

APPOINTMENTS.

THE undermentioned appointments, changes *et cetera* have been promulgated in the *Commonwealth of Australia Gazette*, Number 66, of September 6, 1951.

NAVAL FORCES OF THE COMMONWEALTH.

Permanent Naval Forces of the Commonwealth (Sea-Going Forces).

Resignation.—The resignation of Geoffrey Derek Banyard of his appointment as Surgeon Lieutenant (for short service) is accepted, dated 16th July, 1951.

Emergency List.

Transfer to the Retired List.—Surgeon Lieutenant-Commander William Langley Brookes is transferred to the Retired List, dated 10th July, 1951.

Citizen Naval Forces of the Commonwealth.

Royal Australian Naval Reserve.

Appointments. — Arthur George Harrold (Surgeon Lieutenant, Royal Australian Naval Volunteer Reserve) is appointed Surgeon Lieutenant, with seniority in rank of 29th November, 1947, dated 6th July, 1951. Aretas William Overton Young (Surgeon Lieutenant, Royal Australian Naval Volunteer Reserve) is appointed Surgeon Lieutenant, with seniority in rank of 21st December, 1947, dated 17th July, 1951.

AUSTRALIAN MILITARY FORCES.

Royal Australian Army Medical Corps.

SX700068 Lieutenant-Colonel L. W. Jeffries, D.S.D., O.B.E., is appointed from the Retired List, relinquishes the rank of Lieutenant-Colonel and to be Captain (Honorary Lieutenant-Colonel), 2nd July, 1951.

Citizen Military Forces.

Southern Command: Third Military District.

Royal Australian Army Medical Corps (Medical).—3/58519 Lieutenant E. J. Wellsted is transferred from Royal Australian Artillery (Light Anti-Aircraft) (2nd Military District) and to be Captain (provisionally), 27th April, 1951.

Central Command: Fourth Military District.

Royal Australian Army Medical Corps (Medical).—4/35205 Major J. McPhie is seconded whilst undergoing post-graduate studies in the United Kingdom, 1st August, 1951.

Tasmanian Command: Sixth Military District.

Royal Australian Army Medical Corps (Medical).—6/5128 Honorary Captain D. B. Nathan is appointed from the Reserve of Officers, and to be Captain (provisionally), 29th June, 1951.

Reserve Citizen Military Forces.

Royal Australian Army Medical Corps.

4th Military District.—The resignation of Honorary Captain B. E. Brookman of his commission is accepted, 16th May, 1951.

ROYAL AUSTRALIAN AIR FORCE.

Permanent Air Force: Medical Branch.

The following are appointed to short-service commissions on probation for a period of twelve months, 12th June, 1951, with the rank of Flight Lieutenant: Aubrey Desmond Litchfield (024304), Max Noel McLaughlin (024303).

DISEASES NOTIFIED IN EACH STATE AND TERRITORY OF AUSTRALIA FOR THE WEEK ENDED AUGUST 25, 1951.[1]

Disease.	New South Wales.	Victoria.	Queensland.	South Australia.	Western Australia.	Tasmania.	Northern Territory.[2]	Australian Capital Territory.	Australia.[3]
Ankylostomiasis	*	..	3	..	..	..	..	..	3
Anthrax	*	..	..	..	..	..	..	..	..
Beriberi	*	*	*	*	*	*	..	*	..
Bilharziasis	*	..	..	..	..	..	..	..	..
Cerebro-spinal Meningitis	13(12)	4(4)	..	1(1)	..	..	..	..	18
Cholera	..	..	..	..	..	..	..	..	..
Coastal Fever(*a*)	*	*	..	*	*	*	*	*	..
Dengue	*	*	*	*	*	*	..	*	..
Diarrhœa (Infantile)	..	..	2(1)	..	..	..	..	..	2
Diphtheria	9	8(4)	1(1)	..	6(6)	..	..	..	24
Dysentery (Amœbic)	*	..	..	..	..	..	..	..	..
Dysentery (Bacillary)	*	1(1)	..	..	1(1)	..	..	..	2
Encephalitis Lethargica	..	1	..	..	..	..	..	..	1
Erysipelas	*	*	*	..	*	*	..	..	..
Filariasis	*	..	..	..	..	..	..	..	..
Helminthiasis	*	..	*	*	*	*	*	*	..
Hydatid	*	..	*	*	..	*	*	..	..
Influenza	*	*	*	..	*	*	..	*	..
Lead Poisoning	*	*	..	*	*	*	*	*	..
Leprosy	..	..	..	..	1	..	..	..	1
Malaria(*b*)	*	..	..	..	..	..	..	..	..
Measles	*	*	*	118(66)	*	*	..	..	118
Plague	..	..	..	..	..	..	..	..	..
Poliomyelitis	23(8)	25(4)	14(4)	38(29)	2(1)	..	..	..	102
Psittacosis	*	..	*	..	*	*	*	*	..
Puerperal Fever	..	..	..	..	..	..	..	..	..
Rubella(*c*)	*	6(2)	..	*	6(1)	*	..	..	12
Scarlet Fever	17(13)	16(13)	6(4)	3(2)	3(2)	..	..	..	45
Smallpox	..	..	..	..	..	..	..	..	..
Tetanus	*	..	..	*	*	*	*	*	..
Trachoma	*	..	*	*	*	*	*	*	..
Tuberculosis(*d*)	49(41)	10(6)	19(14)	8(5)	14(10)	..	..	..	100
Typhoid Fever(*e*)	..	1(1)	..	..	1	..	..	..	2
Typhus (Endemic)(*f*)	..	..	1	..	1	..	..	..	2
Undulant Fever	1	..	..	..	..	..	..	..	1
Weil's Disease(*g*)	*	*	..	*	*	*	*	*	..
Whooping Cough	*	*	*	..	*	*	..	..	..
Yellow Fever	..	..	..	..	..	..	..	..	..

[1] The form of this table is taken from the *Official Year Book of the Commonwealth of Australia*, Number 37, 1946–1947. Figures in parentheses are those for the metropolitan area.
[2] Figures not available.
[3] Figures incomplete owing to absence of returns from the Northern Territory.
* Not notifiable.
(*a*) Includes Mossman and Sarina fevers. (*b*) Mainly relapses among servicemen infected overseas. (*c*) Notifiable disease in Queensland in females aged over fourteen years. (*d*) Includes all forms. (*e*) Includes enteric fever, paratyphoid fevers and other Salmonella infections. (*f*) Includes scrub, murine and tick typhus. (*g*) Includes leptospiroses, Weil's and para-Weil's disease.

Rudolph Douglas Grahame Vann (024187) is appointed to a short-service commission, on probation for a period of twelve months, 5th May, 1951, with the rank of Squadron Leader.

The resignation of Squadron Leader J. W. L. Atkinson (034056) is accepted, 18th June, 1951.

Air Force Reserve: Medical Branch.

Flying Officer N. A. Bridgefoot (0210481) is transferred from the General Duties Branch, 5th May, 1951.

John Lister Colless Lahz, M.B., B.S. (257817) is appointed to a commission with the rank of Flight Lieutenant, 14th May, 1951.

Australian Medical Board Proceedings.

QUEENSLAND.

THE undermentioned have been registered, pursuant to the provisions of *The Medical Acts*, 1939-1948, of Queensland, as duly qualified medical practitioners:

Mathieson, John Milton, M.B., B.S., 1948 (Univ. Sydney), P.O. Box 7, Mungindi, New South Wales.

Mason, Charles Edward Alan, M.R.C.S. (England), L.R.C.P. (London), 1949, c.o. Dr. J. Woodley, Prospect Terrace, Hamilton, Brisbane.

Locke, Gerald Macmillan, M.B., B.S., 1951 (Univ. Sydney), Maryborough Base Hospital, Maryborough.

Carmichael, Lindsay Gordon, M.B., Ch.B., 1927 (Univ. Edinburgh), Margate.

The following additional qualifications have been registered:

Saxton, William John, M.C.R. (Aust. and N.Z.), 1950, Inchcolm, Wickham Terrace, Brisbane.

Nye, John Atherton, M.R.A.C.P., 1951, Aroonbeta, Hipwood Street, Hamilton, Brisbane.

Murphy, Clement John, D.O., R.C.O.G., 1950, 158 Gympie Road, Kedron, Brisbane.

Notice.

THE ARTHUR WILSON MEMORIAL FUND.

THE following donations have been received for the Arthur Wilson Memorial Fund, which will be devoted to research into problems of childbirth; they are acknowledged with thanks. Donations may be sent to Dr. C. K. Churches, honorary treasurer, 122 Flinders Street, Melbourne, C.1, and will be acknowledged in this journal. Previously acknowledged £2205 5s., Mr. Leo Doyle £10 10s., Mr. and Mrs. Charles Littlejohn £10 10s., Dr. P. H. Davis £5 5s., Dr. A. Linley Hare £5 5s., Dr. Eileen M. Murphy £5 5s., Dr. Younger Ross £5 5s., Dr. J. A. H. Sherwin £2 2s. Total £2249 7s.

Medical Appointments.

Dr. D. J. Stanger has been appointed government medical officer at Texas, Queensland.

Dr. G. A. W. Pryor has been appointed a member and the chairman and Dr. J. H. Playne and Dr. B. B. Joyce have been appointed members of the medical board under the provisions of *The Workers' Compensation (Lead Poisoning, Mount Isa) Acts*, 1933 *to* 1945, Queensland.

Nominations and Elections.

THE undermentioned have applied for election as members of the New South Wales Branch of the British Medical Association:

Findlay, Wilmet Helena, M.B., B.S., 1951 (Univ. Sydney), Rachel Forster Hospital for Women and Children, Redfern.

Ireland, Basil John, M.B., B.S., 1950 (Univ. Sydney), Saint Vincent's Hospital, Darlinghurst.

The undermentioned have been elected members of the New South Wales Branch of the British Medical Association:

Brown, John Fergus Francis, M.B., B.S., 1948 (Univ. Sydney), c.o. Public Health Department, Madang, T.N.G.

Higgins, Ivy Patricia, M.B., B.S., 1951 (Univ. Sydney), Broken Hill and District Hospital, Broken Hill.

Sheehan, Anthony Hawthorne, M.B., B.S., 1951 (Univ. Sydney), 21 Alexandra Street, Hunter's Hill.

Shellshear, Stuart Wilton, M.B., B.S., 1951 (Univ. Sydney), The Maitland Hospital, Maitland.

Grossy, Moses, registered in accordance with Section 17 (1) (*c*) of *Medical Practitioners Act*, 1938-1950, 15 Plowman Street, North Bondi.

Diary for the Month.

SEPT. 25.—New South Wales Branch, B.M.A.: Ethics Committee.
SEPT. 26.—Victorian Branch, B.M.A.: Council Meeting.
SEPT. 27.—New South Wales Branch, B.M.A.: Branch Meeting.
SEPT. 28.—New South Wales Branch, B.M.A.: Annual (1951) Meeting of Delegates.
SEPT. 28.—Queensland Branch, B.M.A.: Council Meeting.

Medical Appointments: Important Notice.

MEDICAL PRACTITIONERS are requested not to apply for any appointment mentioned below without having first communicated with the Honorary Secretary of the Branch concerned, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

New South Wales Branch (Medical Secretary, 135 Macquarie Street, Sydney): All contract practice appointments in New South Wales.

Victorian Branch (Honorary Secretary, Medical Society Hall, East Melbourne): Associated Medical Services Limited; all Institutes or Medical Dispensaries; Australian Prudential Association, Proprietary, Limited; Federal Mutual Medical Benefit Society; Mutual National Provident Club; National Provident Association; Hospital or other appointments outside Victoria.

Queensland Branch (Honorary Secretary, B.M.A. House, 225 Wickham Terrace, Brisbane, B17): Brisbane Associated Friendly Societies' Medical Institute; Bundaberg Medical Institute. Members accepting LODGE appointments and those desiring to accept appointments to any COUNTRY HOSPITAL or position outside Australia are advised, in their own interests, to submit a copy of their Agreement to the Council before signing.

South Australian Branch (Honorary Secretary, 178 North Terrace, Adelaide): All Lodge appointments in South Australia; all Contract Practice appointments in South Australia.

Western Australian Branch (Honorary Secretary, 205 Saint George's Terrace, Perth): Norseman Hospital; all Contract Practice appointments in Western Australia. All government appointments with the exception of those of the Department of Public Health.

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